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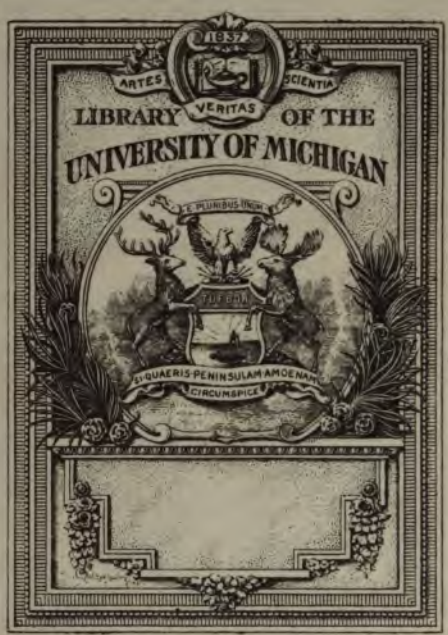
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OCTOBER, 1883.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *ABERCROMBIE, JOHN, M.D., Physician to the Cheltenham General Hospital; 13, Suffolk square, Cheltenham.

1877 ABERCROMBIE, JOHN, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 39, Welbeck street, Cavendish square.

1851 *ACLAND, HENRY WENTWORTH, C.B., M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

1852 †ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for the Paralysed and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, M.A. and M.D., F.R.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.

1879 ALLCHIN, WILLIAM HENRY, M.B., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 5, Chandos street, Cavendish square, W.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's park; 48, Harley street, Cavendish square. *Trans.* 2.

1881 ANDERSON, JAMES, A.M., M.D., 84, Wimpole street, Cavendish square.

1862 ANDREW, EDWYN, M.D., Hardwick House, St. John's Hill, Shrewsbury.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. *Trans.* 1.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1880 *APPLETON, HENRY, M.D., Staines.

1878 ARNOLD, JOHN, Medical Officer of Health; Trinidad.

1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire, and 36, Sussex gardens, Hyde park. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Referee*, 1842-45, 1849-54, 1857-64. *Lib. Com.* 1836. *Trans.* 8.

Elected

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital; 3, Waterloo street, Birmingham.

1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.

1865 BAKER, WILLIAM MORRANT, Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. C. 1878-9. *Lib. Com.* 1876-7. *Trans.* 6.

1869 BAKEWELL, ROBERT HALL, M.D., Ross, Westland, New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Lib. Com.* 1849. *Trans.* 2.

1848 †BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

1866 *BANKS, JOHN THOMAS, M.D., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to the Coombe Hospital; Member of the Senate of the Queen's University in Ireland; 11, Merrion square east, Dublin.

1847 †BARCLAY, ANDREW WHYTE, M.D., Consulting Physician to St. George's Hospital; Medical Officer of Health for Chelsea; Whitney Wood, Stevenage. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. P. 1881. *Referee*, 1863-4, 1870-1, 1877-80. *Trans.* 2.

1879 BARKER, ARTHUR EDWARD JAMES, Assistant Surgeon to, and Assistant Professor of Clinical Surgery at, University College Hospital; 87, Harley street, Cavendish square. *Trans.* 3.

Elected

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service [care of ARTHUR E. J. BARKER, 87, Harley street].

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 109, Gloucester place, Portman square. C. 1844-5. V.P. 1853-4. T. 1860-2. *Referee*, 1848-51. *Trans.* 6.

1876 BARLOW, THOMAS, M.D. and B.S. Lond., Assistant Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond Street; 10, Montague street, Russell square. *Trans.* 1.

1881 *BARNES, HENRY, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 15, Harley street, Cavendish square. C. 1877-8. *Referee*, 1867-76. *Lib. Com.* 1869-73. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Bayswater.

1880 BARROW, A. BOYCE, Assistant Surgeon to the Westminster Hospital; 17, Welbeck street, Cavendish square, W.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, *Vice-President*, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 32, George street, Hanover square. C. 1876-77. V.P. 1883. *Referee*, 1868-75, 1879-82. *Trans.* 9.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 20, Queen Anne street, Cavendish square. *Trans.* 1.

1874 BAXTER, EVAN BUCHANAN, M.D., Professor of Materia Medica at King's College, London; Physician to King's College Hospital; Examiner in Materia Medica at the University of London; 28, Weymouth street, Portland place. *Referee*, 1881-3.

Elected

1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Assistant Physician to the City of London Hospital for Diseases of the Chest; and Physician to the Great Northern Hospital; 23, Upper Berkeley street.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

1860 *BEALEY, ADAM, M.D., M.A. Camb., Oak Lea, Harrogate.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871 BECK, MARCUS, M.S., Teacher of Operative Surgery, and Assistant Professor of Clinical Surgery in University College, London; and Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. *Referee*, 1882-3. *Lib. Com.* 1881-3.

1880 BEEVOR, CHARLES EDWARD, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 129, Harley street, Cavendish square.

1858 BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell; 26, Saint Peter's square, Hammersmith. C. 1877–8.

1883 BELL, HUTCHINSON ROYES, Surgeon to, and Demonstrator of Operative Surgery at, King's College Hospital; 12, Queen Anne street, Cavendish square.

1871 BELLAMY, EDWARD, Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Lecturer on Artistic Anatomy to the Science and Art Department, South Kensington; 17, Wimpole street, Cavendish square. *Referee*, 1882-3. *Lib. Com.* 1879-81.

1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.

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1880 BENNETT, ALEX. HUGHES, M.D., Assistant Physician to the Westminster Hospital; 38, Queen Anne street, Cavendish square, and 1, Petersham terrace, Gloucester road, South Kensington.

1877 BENNETT, WILLIAM HENRY, Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; Surgeon to the Belgrave Hospital for Children; 1, Chesterfield street, Mayfair.

1845 †BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1878 BINDON, WILLIAM JOHN VEREKER, M.D.

1854 BIRD, PETER HINCKES, F.L.S., Post Office, Limassol, Cyprus.

1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 †BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6. *Referee*, 1851-9.

1851 †BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1866 BISHOP, EDWARD, M.D., Cintra park, Upper Norwood.

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1881 Biss, Cecil Yates, M.B., Assistant Physician to, and Lecturer on Botany at, the Middlesex Hospital; Assistant Physician to the Hospital for Consumption; 65, Harley street, Cavendish square.

1865 Blanchet, Hilarion, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 Blandford, George Fielding, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street. C. 1883.

1867 Bloxam, John Astley, Surgeon to, and Teacher of Operative Surgery in, Charing Cross Hospital; Surgeon for Out-Patients to the Lock Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 Bojanus, Louis Henry, M.D., Wilna.

1846 †Bostock, John Ashton, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 73, Onslow gardens, Brompton. C.1861-2. V.P. 1870-71. *Sci. Com.* 1867.

1869 Bourne, Walter, M.D. [care of the National Bank of India, 80, King William street, City.]

1882 Bowlby, Anthony A., Curator of the Museum, St. Bartholomew's Hospital, 75, Warrington crescent, Maida hill.

1870 *Bowles, Robert Leamon, M.D., 8, West terrace, Folkestone.

1841 †Bowman, William, LL.D., F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Referee*, 1845-50, 1854-6. *Lib. Com.* 1847. *Trans.* 3.

1862 Brace, William Henry, M.D., 7, Queen's Gate terrace, Kensington.

Elected

1874 BRADSHAW, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1867 *BRETT, ALFRED T., M.D., Watford, Herts.

1876 BRIDGES, ROBERT, M.B., Manor House, Yattendon, Berks.

1867 BRIDGEWATER, THOMAS, M.B. Lond., Harrow-on-the-Hill, Middlesex.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Referee*, 1881-3. *Trans.* 3.

1851 †BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 3, Chesterfield street, Mayfair. *Trans.* 1.

1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., LL.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1862.

1878 BROWNE, JAMES CRICHTON, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 7, Cumberland Terrace, Regent's Park.

1880 BROWNE, JAMES WILLIAM, M.B., 8, Norland place, Uxbridge road.

1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Ophthalmological Hospital; 10, College square N., Belfast.

1881 BROWNE, OSWALD A., M.A., St. Bartholomew's Hospital; 25, Bernard Street, Russell Square.

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley street. *Trans.* 1.

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1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; 50, Welbeck street, Cavendish square. *Referee*, 1880-83. *Lib. Com.* 1882-3.

1860 BRYANT, THOMAS, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Sci. Com.* 1863. *Referee*, 1882-3. *Lib. Com.* 1868-71. *Trans.* 9. *Pro.* 1.

1855 BRYANT, WALTER JOHN, M.R.C.P. Edinb.; Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., F.R.S., Medical Officer of the Local Government Board; Member of the Senate of the University of London; 24, Nottingham place, Marylebone road.

1864 BUCKLE, FLEETWOOD, M.D.

1876 BUCKNILL, JOHN CHARLES, M.D., F.R.S.; E 2, The Albany, Piccadilly, and Hill Morton Hall, Rugby,

1881 BULLER, AUDLEY CECIL, City of London Hospital for Diseases of the Chest, Victoria Park; 10, Grafton Street, Bond Street.

1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Referee*, 1842-6, 1850-7, 1861-68, 1875-81. *Lib. Com.* 1836. *Trans.* 2.

1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Referee*, 1846-54, 1857-65. *Lib. Com.* 1847. *Trans.* 4.

Elected

1873 BUTLIN, HENRY TRENTHAM, Assistant Surgeon to, and Demonstrator of Practical Surgery and of Diseases of the Larynx at, St. Bartholomew's Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 3.

1871 BUTT, WILLIAM F., 25, Park street, Park lane.

1883 BUXTON, DUDLEY WILMOT, M.B., B.S., 99, Gower Street.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde park.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.

1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.* 1863.

1879 CARTWRIGHT, S. HAMILTON, Professor of Dental Surgery at King's College; 32, Old Burlington street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square.

1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

Elected

1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital; Consulting Physician to the Lock Hospital; Shrubs Hill House, Sunningdale, Staines. C. 1861. V.P. 1867. L. 1869-72. *Referee*, 1851-60, 1866. *Lib. Com.* 1852, 1868. *Trans.* 1.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.B., Assistant Obstetric Physician to St. George's Hospital; 60, Great Cumberland place *Trans.* 3.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; Crofton Lodge, Hopton road, Coventry park, Streatham.

1881 *CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the General Hospital, Birmingham; 108, New Hall street, Birmingham.

1868 CHEADLE, WALTER BUTLER, M.D., Physician (with charge of out-patients) to, and Lecturer on Medicine at, St. Mary's Hospital; Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.

1879 CHEYNE, WILLIAM WATSON, M.B., Assistant Surgeon and Demonstrator of Surgery to King's College Hospital; 14, Mandeville place, Manchester square, W.

1873 *CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.

1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.

Elected

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. *Referee* 1874-81.

1860 CLARK, SIR ANDREW, Bart., M.D., LL.D., Aberd., Physician to, and Lecturer on Medicine at, the London Hospital; 16, Cavendish square. C. 1875.

1879 CLARK, ANDREW, Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 19, Cavendish place, Cavendish square, W.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Referee*, 1859-81. *Lib. Com.* 1847. *Trans.* 5.

1882 CLARKE, ERNEST, M.B., B.S., late Assistant Chloroformist to St. Bartholomew's Hospital; 6, Lee Terrace, Blackheath.

1848 †CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.

1881 CLARKE, W. BRUCE, M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 46, Harley street, Cavendish square.

1866 CLARKE, WILLIAM FAIRLIE, M.D., M.A. Oxon., Southborough, Tunbridge Wells. *Trans.* 2.

1842 †CLAYTON, SIR OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

1879 CLUTTON, HENRY HUGH, M.A., M.B., Assistant Surgeon to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 77, Lambeth Palace road.

1857 COATES, CHARLES, M.D., Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 13, Spring gardens, Charing cross. *Trans.* 2.

Elected

1865 COOPER, ALFRED, Surgeon to the Lock Hospital; Assistant Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square.

1843 †COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.P. 1873-4. *Lib. Com.* 1847, 1856-7.

1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 146, Donegall Pass, Belfast.

1853 CORY, WILLIAM GILLETT, M.D., 1, Caledonia place, Clifton, Bristol.

1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.

1860 †COUPER, JOHN, Surgeon to the London Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876. *Referee* 1882-3.

1877 COUPLAND, SIDNEY, M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 14, Weymouth street, Portland place.

1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Millwood, Wilbury road, Brighton. C. 1853-4.

1868 CRAWFORD, THOMAS, M.D., Director General, Army Medical Department; 6, Whitehall yard, and 5, St. John's park, Blackheath.

1873 CREIGHTON, CHARLES, M.D., 6, Queen Anne street, Cavendish square. *Referee*, 1882-3. *Trans.* 1.

Elected

1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

1874 CRIPPS, WILLIAM HARRISON, Assistant Surgeon to St. Bartholomew's Hospital; Surgeon to the Great Northern Hospital; Assistant Surgeon to the Royal Free Hospital; 2, Stratford place, Oxford street. *Trans.* 1.

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; Assistant Physician to the East London Hospital for Children; 28, Welbeck Street, Cavendish Square.

1868 CROFT, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Thomas's Hospital; 48, Brook street, Grosvenor square. *Lib. Com.* 1877-8. *Trans.* 1.

1862 CROMPTON, SAMUEL, M.D., late Physician to the Salford Royal Hospital and Dispensary; Brookmead, Cranleigh, Surrey.

1837 CROOKES, JOHN FARRAR, 45, Augusta gardens, Folkestone.

1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.

1879 CUMBERBATCH, A. ELKIN, Aural Surgeon to St. Bartholomew's Hospital; Aural Surgeon to the Great Northern Hospital; 17, Queen Anne street.

1846 CURLING, HENRY, Consulting Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 27, Brunswick square, Brighton. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Referee*, 1844-6, 1851-3, 1858, 1865-70, 1875-9. *Sci. Com.* 1863. *Lib. Com.* 1839. *Trans.* 13. *Pro.* 1.

1873 CURNOW, JOHN, M.D., Professor of Anatomy at King's College, London, and Physician to King's College Hospital; 3, George street, Hanover square.

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

Elected

1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 2.

1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.

1877 DARBISHIRE, SAMUEL DUKINFIELD, M.B., Physician to the Radcliffe Infirmary, Oxford.

1879 DARWIN, FRANCIS, M.B., F.R.S., Down, Bromley, Kent.

1848 DAUBENY, HENRY, M.D., San Remo, Italy.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 2, Gambier terrace, Liverpool.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 2, Marlborough buildings, Bath.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 2.

1878 DAVY, RICHARD, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square.

1882 DAWSON, YELVERTON, M.D., 28, Hyde park street.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1878 DENT, CLINTON THOMAS, Assistant Surgeon to St. George's Hospital; 19, Savile row, Burlington gardens. *Trans.* 2.

1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.

1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at St. George's Hospital, and Senior Physician to the Hospital for Sick Children, Examiner in Medicine at the University of London; 9, Chesterfield street, Mayfair. C. 1874-5. *Referee*, 1869-73, 1882-3. *Sci. Com.* 1867-79 *Trans.* 13.

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Consulting Ophthalmic Surgeon to the Asylum for Idiots; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

Elected

1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; Streate place, Bournemouth. *Trans.* 2.

1845 DODD, JOHN.

1879 DONKIN, HORATIO, M.B., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 60, Upper Berkeley street, Portman square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to Out-Patients, Samaritan Hospital; 51, Seymour street, Portman square.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 81, Harley street, Cavendish square. C. 1880. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1879 DREWITT, F. G. DAWTREY, M.D., Assistant Physician to the West London Hospital and to the Victoria Hospital for Children; 52, Brook street, Grosvenor square.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 65, Regent street, W.

1865 DUCKWORTH, DYCE, M.D., Physician to St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883. *Trans.* 1.

1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 125, Cromwell road, South Kensington.

1845 DUFF, GEORGE, M.D., High street, Elgin.

1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.

1874 DUKA, THEODORE, M.D., [Surgeon-Major, H.M.'s Bengal Army]; Torquay, Devonshire.

Elected

1871 DUKE, BENJAMIN, 2, Windmill road, Clapham common.

1871 *DUKES, CLEMENT, M.D. and B.S., Horton crescent, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Argyle House, Clapham common.

1877 DUNCAN, JAMES MATTHEWS, M.D., LL.D. Ed., F.R.S. Ed., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; Examiner in Obstetric Medicine, University of London; 71, Brook street, Grosvenor square. *Referee*, 1881-3.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 DURHAM, FREDERIC, M.B., 38, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1836 EARLE, JAMES WILLIAM, late of Norwich.

1868 EASTES, GEORGE, M.B. Lond., Surgeon-Accoucheur to the Western General Dispensary; 69, Connaught street, Hyde park square.

1883 EDMUNDS, WALTER, M.D., 79, Palace Road, Albert Embankment.

1883 EDWARDES, EDWARD JOSHUA, M.D., 17, Orchard street, Portman Square, W.

1824 EDWARDS, GEORGE.

1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.

1869 ELAM, CHARLES, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 75, Harley street, Cavendish square.

Elected

1848 ELLIS, GEORGE VINER, late Professor of Anatomy in University College, London; Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1842 †ERICHSEN, JOHN ERIC, F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery in University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish sq. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-7. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Pathological Curator of the Museum, Royal College of Surgeons; Surgical Registrar to St. Bartholomew's Hospital; 14, Furnival's Inn, Holborn. *Trans.* 2.

1877 EWART, WILLIAM, M.B., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital; 33, Curzon street, Mayfair.

1875 *FAGAN, JOHN, Surgeon to the Belfast Hospital for Sick Children; 11, College square north, Belfast.

1864 †FAGGE, CHARLES HILTON, M.D., Physician to, and Lecturer on Pathology at, Guy's Hospital; Examiner in Medicine at the University of London; 76, Grosvenor street. C. 1880-81. L. 1883. *Referee*, 1874-9. *Sci. Com.* 1879. *Lib. Com.* 1875-9. *Trans.* 7.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.

1862 FARQUHARSON, ROBERT, M.D., M.P., Finzean, Aboyne, Aberdeenshire, and the Reform Club, Pall Mall. *Lib. Com.* 1876-80.

Elected

1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 18, Albert Mansions, Victoria street, Westminster. C. 1857. V.P. 1864. *Referee*, 1848-54, 1861-3, 1865-6. *Sci. Com.* 1863. *Lib. Com.* 1847.

1872 FAYRER, SIR JOSEPH, K.C.S.I., M.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-General, late Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 53, Wimpole street, Cavendish square. *Referee*, 1881-3.

1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 16, Old Elvet, Durham.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882-3. *Trans.* 4.

1880 FERRIER, DAVID, M.D., F.R.S., Professor of Forensic Medicine at King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 16, Upper Berkeley street, Portman square, W.

1852 *FIELD, ALFRED GEORGE.

1849 †FINCHAM, GEORGE TUPMAN, M.D., Consulting Physician to the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

1879 FINLAY, DAVID WHITE, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Physician to the Royal Hospital for Diseases of the Chest; 21, Montagu street, Portman square.

1866 FISH, JOHN CROCKETT, B.A., M.D. Camb., Assistant Physician to the West London Hospital; 92, Wimpole street, Cavendish square.

1866 FITZPATRICK, THOMAS, M.D., M.A., Dublin; Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.

Elected

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 43, Clarendon square, Leamington. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1877 FONMARTIN, HENRY DE, M.D., Knaphill, Woking, Surrey.

1848 †FORBES, JOHN GREGORY, Egerton House, Egerton, Ashford, Kent. C. 1868-9. *Lib. Com.* 1855. *Trans.* 2.

1852 †FORSTER, JOHN COOPER, *Treasurer*, Consulting Surgeon to the Royal Hospital for Children and Women; Vice-President of the Royal College of Surgeons; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. V.P. 1877-8. T. 1879-83. *Referee*, 1870-2, 1876. *Pro.* 1.

1877 *FORTESCUE, GEORGE, M.B., late Surgeon to the Sydney Infirmary; 6, Lyons terrace, Sydney, New South Wales.

1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.

1883 FOWLER, JAMES KINGSTON, M.B., Assistant Physician to the Middlesex Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine; Church House, Clifton, Gloucestershire.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician to the Skin Department of the North-west London Hospital, and Assistant Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

Elected

1858 Fox, Wilson, M.D., F.R.S., *Librarian*, Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 67, Grosvenor street. C. 1875-6. L. 1883. *Referee*, 1869-74. *Lib. Com.* 1866-70, 1874. *Trans.* 3.

1871 Frank, Philip, M.D., Cannes, France.

1843 Fraser, Patrick, M.D. C. 1866.

1868 Freeman, William Henry, 21, St. George's square, South Belgravia.

1836 †French, John George, 10, Cunningham place, Maida hill. C. 1852-3.

1883 Fuller, Henry Roxburgh, M.B., 1, St. George's place, Hyde park.

1876 Furner, Willoughby, 111, King's road, Brighton.

1864 *Gairdner, William Tennant, M.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 225, St. Vincent street, Glasgow.

1874 Galabin, Alfred Lewis, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant Physician to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Referee*, 1882-3. *Lib. Com.* 1883. *Trans.* 2.

1883 Galton, John Charles, M.A., F.L.S., 45, Great Marlborough street, W.

1865 Gant, Frederick James, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde park. C. 1880-81. *Lib. Com.* 1882-3. *Trans.* 3.

1867 Garland, Edward Charles, L.R.C.P. Edin., Yeovil, Somerset.

1867 Garlike, Thomas W., Malvern Villa, Churchfield road, Ealing.

Elected

1854 GARROD, ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

1879 GARSTANG, THOMAS WALTER HARROPP, Oakleigh, Dobcross, near Manchester.

1851 †GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park. C. 1875-6. *Trans.* 2.

1819 GAULTER, HENRY.

1848 †GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 34, Finsbury place. C. 1874-5.

1866 GEE, SAMUEL JONES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Physician to the Hospital for Sick Children; 54, Wimpole street, Cavendish square. C. 1883. *Sci. Com.* 1879. *Lib. Com.* 1871-6. *Trans.* 1.

1878 GERVIS, HENRY, M.D., Obstetric Physician to, and Lecturer on Obstetric Medicine at, St. Thomas's Hospital; Examiner in Obstetric Medicine at the University of London; 40, Harley street, Cavendish square.

1880 GIBBONS, ROBERT ALEXANDER, M.D., 32, Cadogan place.

1877 GODLEE, RICKMAN JOHN, Assistant-Surgeon to University College Hospital; and Demonstrator of Anatomy in University College; 81, Wimpole street, Cavendish square.

1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; 9, Grosvenor street, Grosvenor square.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

Elected

1883 GOODHART, JAMES FREDERIC, M.D., Assistant Physician to, and Curator of the Museum at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; Surgeon to the North-west London Hospital; 16, Queen Anne street, Cavendish square.

1873 GOWERS, WILLIAM RICHARD, M.D., Assistant Professor of Clinical Medicine in University College, and Assistant-Physician to University College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. *Trans.* 6.

1851 †GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Crawleydown park, Worth, Sussex. C. 1863.

1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. *Referee*, 1882-3.

1875 *GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1843 †GREENHALGH, ROBERT, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 35, Cavendish square. C. 1871-2. *Referee*, 1876-7. *Trans.* 1.

1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; Castle Lodge, Reigate. C. 1876-7. *Referee*, 1870-5. *Trans.* 3.

Elected

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; Assistant-Physician to the Victoria Hospital for Children; 6, Curzon street, Mayfair.

1852 GROVE, JOHN, Westgate court, Canterbury.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris. *Lib. Com.* 1863-5.

1849 †GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Referee*, 1855-63. *Trans.* 4.

1854 HABERSHON, SAMUEL OSBORNE, M.D., 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. V.P. 1881-2. *Referee*, 1862-6, 1868, 1871-80. *Trans.* 3.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Assistant Physician, and Physician to the Throat Department, and Lecturer on Forensic Medicine at the Westminster Hospital; Physician to St. Mark's Hospital; 46, Queen Anne street, Cavendish square.

1870 HAMILTON, ROBERT, Surgeon to the South Hospital, Liverpool; 1 Prince's road, Liverpool.

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; Florence road, Ealing, and Duff House, Banff, N.B.

1836 HARDING, JOHN FOSSE, Eagle House, Hornsey. C. 1858-9.

1856 HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and late Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4.

1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3. *Trans.* 1.

Elected

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882-3. *Sci. Com.* 1879. *Trans.* 8.

1866 HARPER, PHILIP H., 30, Cambridge street, Hyde park.

1859 HARRIS, FRANCIS, M.D., F.L.S., 24, Cavendish square.

1880 HARRIS, VINCENT DORMER, M.D., Casualty Physician to St. Bartholomew's Hospital, and Assistant Physician to the Victoria Park Hospital; 39, Wimpole street, Cavendish square.

1872 HARRIS, WILLIAM H., M.D., Deputy Surgeon-General, Madras Army (retired); late Professor of Midwifery and Diseases of Women and Children, Madras Medical College.

1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool. *Trans.* 1.

1854 HAVILAND, ALFRED.

1870 HAWARD, J. WARRINGTON, Surgeon to St. George's Hospital; Surgeon to the Hospital for Sick Children; 16, Savile row, Burlington gardens. *Lib. Com.* 1881–3. *Trans.* 1.

1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Referee*, 1843-7, 1850-4, 1857-9, 1861-83. *Trans.* 12.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.

1848 †HAWKSLEY, THOMAS, M.D., Consulting Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 31, Grosvenor street.

Elected

1875 HAYES, THOMAS CRAWFORD, M.D., Assistant-Physician-Accoucheur and Assistant-Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1861 HAYWARD, WILLIAM HENRY, Chapel Ash, Wolverhampton.

1848 *HEALE, JAMES NEWTON, M.D.

1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. *Lib. Com.* 1870-3. *Trans.* 3.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1882 HENSLEY, PHILIP J., M.D., Assistant Physician to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 7, West street, Finsbury circus.

1877 HERON, GEORGE ALLAN, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Assistant Physician to the West London Hospital for the Paralysed and Epileptic; 40, Margaret street, Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.B. Oxon., 22, Bedford square.

1843 HEWETT, SIR PRESCOTT GARDNER, Bart., F.R.S., Serjeant-Surgeon-Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Corresponding Member of the Academy of Medicine, and of the "Société de Chirurgie," Paris; Chesnut Lodge, Horsham, Sussex. C. 1859. V.P. 1866-7. *Referee*, 1850-8, 1860-5, 1868-83. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 7.

Elected

1855 HEWITT, W. M. GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-83. *Lib. Com.* 1868, 1874.

1880 HICKS, CHARLES CYRIL, M.D., Wokingham, Berks.

1873 HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1862 HILL, M. BERKELEY, M.B. Lond., *Secretary*, Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 55, Wimpole street, Cavendish square. C. 1878-9. S. 1881-3. *Trans.* 1.

1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.

1859 HIRD, FRANCIS, Consulting Surgeon to the Charing Cross Hospital; 13, Old Burlington street.

1861 *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

1843 †HOLDEN, LUTHER, Consulting Surgeon to St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1879 HOLLAND, PHILIP ALEXANDER, M.A., Swancoe Park, Macclesfield.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.B., Camb., Assistant-Physician to the Sussex County Hospital; Park Gate, Preston road, Brighton.

1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road, South Hampstead.

Elected

1856 HOLMES, TIMOTHY, M.A. Camb., Surgeon to St. George's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. *Referee*, 1866-8, 1872, 1883. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 8.

1846 †HOLT, BARNARD WIGHT, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3. V.P. 1879-80.

1846 †HOLTHOUSE, CARSTEN, 35, Essex street, Strand, W.C. C. 1863. *Referee* 1870-6. *Lib. Com.* 1859-60.

1878 HOOD, DONALD WILLIAM CHARLES, M.D. Cantab., Assistant Physician to the West London Hospital; 43, Green street, Park lane.

1883 HORSLEY, VICTOR A., Assistant Professor of Pathological Anatomy, University College; 129, Gower Street.

1878 HOUGHTON, WALTER B., M.D., Church Villa, Warrior square, St. Leonards-on-Sea.

1865 HOWARD, BENJAMIN, M.D., New York, U.S.

1865 HOWARD, EDWARD, M.D.

1881 HOWARD, HENRY, M.B., Stockwell Fever Hospital.

1874 HOWSE, HENRY GREENWAY, M.S. Lond., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; Examiner in Anatomy in the University of London; 10, St. Thomas's street, Southwark. *Sci. Com.* 1879. *Trans.* 2.

1877 *HUDSON, ROBERT SAMUEL, M.D., 58, West-end, Redruth, Cornwall.

1857 HULKE, JOHN WHITAKER, F.R.S., *Librarian*, Surgeon to the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-83. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 8.

1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.

Elected

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the Cambridge University Medical School; Cambridge. *Trans.* 6.

1882 HUMPHRY, LAURENCE, M.B., Cambridge.

1873 HUNTER, WILLIAM GUYER, M.D., Hon. Surgeon to H.M. the Queen; late Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-General Bombay Army; 21, Norfolk crescent, Hyde park.

1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81, 1883. *Lib. Com.* 1864-5. *Trans.* 10. *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 †HUTTON, CHARLES, M.D., Consulting Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1847 IMAGE, WILLIAM EDMUND, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., Cairo. [Athenæum Club, Pall Mall.]

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

1841 †JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; 47, Waterloo road, south, Wolverhampton.

Elected

1841 JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.

1883 JACOBSON, WALTER HAMILTON ACLAND, B.A., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 41, Finsbury square.

1825 JAMES, JOHN B., M.D.

1851 †JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1881 JENNINGS, WILLIAM OSCAR, M.D., 8, Rue Roy, Paris.

1883 JESSOP, WALTER H. H., M.B., Assistant Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley Street.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1847 †JOHNSON, GEORGE, M.D., F.R.S., Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib.Com.* 1860-1. *Trans.* 10.

1881 JOHNSON, GEORGE LINDSAY, M.A., M.B., Fern Lea, Highfield hill, Upper Norwood.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Brighton Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1876 JONES, LESLIE, M.D., Medical Officer of Health for Blackpool; 3, Brighton Parade, Blackpool, Lancashire.

Elected

1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]

1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 22, Colmore row, Birmingham.

1881 JULER, HENRY EDWARD, Assistant Surgeon Royal Westminster Ophthalmic Hospital; Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole street Cavendish square.

1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1882 KEETLEY, CHARLES R. B., Assistant Surgeon to the West London Hospital; 20, Princes street, Hanover square.

1872 KELLY, CHARLES, M.D., Professor of Hygiene at King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District, Worthing, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

1877 *KHORY, RUSTONJEE NASERWANJEE, M.D. Brussels; Physician to the Parell Dispensary, Bombay; Lecturer to Native Midwives, Grant Medical College, Bombay [39, St. James's square, Holland park.]

1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater.

1881 KIDD, PERCY, M.A., M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook street, Grosvenor square. *Trans.* 2.

1882 KING, DAVID ALEXANDER, M.B., Assistant Physician to the Hospital for Consumption, Brompton; 26, Harley street, Cavendish square.

Elected

1851 †KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; Greenford, Middlesex. C. 1849-50. V.P. 1865. *Referee*, 1850.

1882 LANG, WILLIAM, Ophthalmic Surgeon to the Middlesex Hospital; 26, Upper Wimpole street, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square. C. 1881-2. *Lib. Com.* 1879-80.

1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1816 LAWRENCE, G. E.

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., 12, Upper Berkeley street, Portman square.

1882 LEDWICH, EDWARD L'ESTRANGE, Lecturer on Surgical and Descriptive Anatomy in the Ledwick School of Medicine, Dublin; 23, Upper Leeson Street, Dublin.

1843 †LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 13. *Pro.* 2.

1877 LEESON, ARTHUR EDMUND, M.A., M.D. [South America.]

1883 LEESON, JOHN RUDD, M.D., C.M., 6, Clifden road, Twickenham.

Elected

1869 LEGG, JOHN WICKHAM, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Bartholomew's Hospital; 47, Green street, Park lane. *Referee*, 1882-3. *Lib. Com.* 1878-83. *Trans.* 2.

1836 LEIGHTON, FREDERICK, M.D.

1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Paris.

1806 LIND, JOHN, M.D.

1878 LISTER, JOSEPH, D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, London; and Surgeon to King's College Hospital; 12, Park crescent, Regent's park.

1872 *LITTLE, DAVID, M.D., Surgeon to the Royal Eye Hospital, Manchester; 21, St. John's street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1881 LOCKWOOD, CHARLES BARRETT, Demonstrator of Anatomy at St. Bartholomew's Hospital: 8, Serjeants' inn, Fleet street.

1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Staines, Surrey.

1877 LOWNE, BENJAMIN THOMPSON, Lecturer on Physiology, Middlesex Hospital Medical School; 65, Cambridge gardens, Notting hill.

Elected

1881 LUCAS, RICHARD CLEMENT, Senior Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury square.

1883 LUND, EDWARD, Professor of Surgery, Owens College; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1857 LYON, FELIX WILLIAM, M.D., 118, Princes street, Edinburgh.

1882 LYONS, ISIDORE, Dental Surgeon to the Evelina Hospital for Children; Assistant Dental Surgeon to St. Bartholomew's Hospital; 19, Queen Anne street, Cavendish square.

1867 MABERLY, GEORGE FREDERICK, 98, Collins street east, Melbourne, Victoria.

1873 MACCARTHY, JEREMIAH, M.A., Surgeon to, and Lecturer on Physiology at, the London Hospital; 15, Finsbury square. *Lib. Com.* 1882-3.

1867 MACCORMAC, SIR WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Examiner in Surgery at the University of London; 13, Harley street. *Trans.* 1.

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 89, Merrion square west, Dublin. *Trans.* 2.

1880 *MACFARLANE, ALEXANDER WILLIAM, M.D., Consulting Physician to the Kilmarnock Fever Hospital and Infirmary; Walmer, Kilmarnock, N.B.

1866 MACGOWAN, ALEXANDER THORBURN, Vyvyan House, Clifton, near Bristol.

1880 MCHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 5, Savile row.

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.

Elected

1873 MacKellar, Alexander Oberlin, M.S.I., Assistant Surgeon to St. Thomas's Hospital; 22, George street, Hanover square.

1881 Mackenzie, Stephen, M.D., Senior Assistant Physician, and Physician in charge of Department of Skin Diseases at the London Hospital; 26, Finsbury sqnare.

1876 Mackey, Edward, M.D., 1, Brunswick road, Hove, Brighton.

1854 *Mackinder, Draper, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1879 Maclagan, Thomas John, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1860 Maclean, John, M.D., 24, Portman street, Portman square.

1876 Macnamara, Charles, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street.

1842 Macnaught, John, M.D., 74, Huskisson street, Liverpool.

1881 Macready, Jonathan F. C. H., Surgical Registrar to St. Bartholomew's Hospital; Surgeon to the Great Northern Hospital; 125, Harley street, Cavendish square.

1880 Maddick, Edmund Distin, 17, Upper Wimpole street.

1880 Makins, George Henry, St. Thomas's Hospital, Albert Embankment.

1876 Mallam, Benjamin, Meadow Side, Leacroft road, Staines.

1855 Marcet, William, M.D., F.R.S.; 39, Grosvenor street. C. 1871. *Referee*, 1866-70, 1883. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 Marsh, F. Howard, Assistant Surgeon to St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children, Great Ormond street; 36, Bruton street, Berkeley square. C. 1882-3. *Lib. Com.* 1880-1. *Trans.* 3.

1838 Marsh, Thomas Parr, M.D.

Elected

1851 †MARSHALL, JOHN, F.R.S., *President*, Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-6. P. 1882-3. *Referee*, 1867, 1871-4, 1877-81. *Trans.* 3.

1864 MASON, FRANCIS, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. C. 1880-81. *Trans.* 1.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square. *Lib. Com.* 1875-7.

1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square, and 11, Montpellier row, Blackheath, Kent.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne street, Cavendish square.

1867 MEREDYTH, COLOMIATI, M.D., 10, George street, Hanover square.

1874 MERRIMAN, JOHN J., 42, Kensington square.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; The Limes, Bristol road, Edgbaston, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1882 MILLS, JOSEPH, 15, Henrietta street, Cavendish square.

1873 MILNER, EDWARD, Surgeon for Out-Patients to the Lock Hospital; 32, New Cavendish street, Portland place.

1883 MONEY, ANGEL, M.D., Hospital for Sick Children, Great Ormond street; 14, Langham place, Regent street. *Trans.* 1.

Elected

1873 MOORE, NORMAN, M.D., Assistant Physician and Warden of the College and Lecturer on Comparative Anatomy, Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. C. 1880-1. *Lib. Com.* 1862-3. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Manchester; 1, St. Peter's square, Manchester.

1878 MORGAN, JOHN HAMMOND, M.A., Assistant Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street; 12, Chapel street, Park lane. *Trans.* 1.

1874 MORRIS, HENRY, M.A. Lond., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Referee*, 1882-3. *Trans.* 7.

1879 MORRIS, MALCOLM ALEXANDER, Lecturer on Skin Diseases at St. Mary's Hospital; 63, Montagu square.

1851 MOUAT, FREDERIC JOHN, M.D., Deputy Inspector-General of Hospitals; Medical Inspector to the Local Government Board; and Member of the Senate of the University of Calcutta.

1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 6, Finsbury Circus. *Referee*, 1879-83. *Trans.* 1.

1879 MUNK, WILLIAM, M.D., Harveian Librarian, Royal College of Physicians; Consulting Physician to the Roya. Hospital for Incurables; 40, Finsbury square.

1875 MURPHY, WILLIAM KIRKPATRICK, M.A., M.D., 29, Queen Anne street, Cavendish square.

1873 MURRAY, IVOR, M.D., F.R.S. Ed., 8, Huntriss Row, Scarborough.

Elected

1880 MURRELL, WILLIAM, M.D., Assistant Physician to the Roya. Hospital for Diseases of the Chest; Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, the Westminster Hospital; 38, Weymouth street, Portland place. *Trans.* 1.

1863 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to the 1st Battalion, Coldstream Guards; Hospital, Vincent square, Westminster. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, A. T., M.D., Medical Registrar, St. George's Hospital; 24, Clarges street, Piccadilly.

1881 NALL, SAMUEL, M.B., Demonstrator of Physiology at St. Mary's Hospital; 34, Highgate road.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park. *Lib. Com.* 1841.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; Ophthalmic Surgeon to the Hospital for Sick Children; 5, Wimpole street, Cavendish square.

1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Duke street, Chelmsford, Essex.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

1847 *NOURSE, WILLIAM EDWARD CHARLES, late Surgeon to the Brighton Children's Hospital; Bouverie House, Mount Radford, Exeter.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

Elected

1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption, and Physician to the Westminster General Dispensary; 11, Welbeck street, Cavendish square.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1880 OGILVIE, GEORGE, M.B., Lecturer on Experimental Physics at the Westminster Hospital; 27, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., Lecturer on Comparative Anatomy at the Westminster Hospital; 46, Welbeck street, Cavendish square.

1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. *Referee*, 1864-72. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; The Elms, Duffield road, Derby.

1860 OGLE, WILLIAM, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. *Lib. Com.* 1871-5. *Trans.* 4.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 7, Brook street, Hanover square. *Trans.* 6.

1877 ORMEROD, JOSEPH ARDERNE, M.B., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 25, Upper Wimpole street.

1875 OSBORN, SAMUEL, 10, Maddox street, Regent street, and 17, Gresham park, Brixton.

1879 OWEN, EDMUND, Surgeon to St. Mary's Hospital; Surgeon to the Hospital for Sick Children; 49, Seymour street, Portman square. *Trans.* 1.

Elected

1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 41, Gloucester gardens, Hyde Park.

1874 PAGE, HERBERT WILLIAM, M.A., M.C. Cantab., Surgeon (with charge of out-patients) to, and Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. *Trans.* 2.

1847 *PAGE, WILLIAM BOUSFIELD, Consulting Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.

1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

1847 PARKER, NICHOLAS, M.D., Paris.

1873 PARKER, ROBERT WILLIAM, Assistant-Surgeon to the East London Hospital for Children; 8, Old Cavendish street. *Trans.* 2.

1841 PARKIN, JOHN, M.D., 5, Codrington place, Brighton.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 35, Grosvenor street. C. 1883. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Assistant-Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. *Sci. Com.* 1879. *Lib. Com.* 1878-83.

1879 PEEL, ROBERT, L.K.Q.C.P.I., 120, Collins street east, Melbourne, Victoria.

1856 PEIRCE, RICHARD KING, Woodside, Windsor forest, Berks.

Elected

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 12, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's Sussex.

1870 PERRIN, JOHN BESWICK, late Medical Tutor and Demonstrator of Practical and Surgical Anatomy, Owen's College; Vernon House, Leigh, Lancashire.

1879 *PESIKAKA, HORMASJI DOSABHAI, L.R.C.P. Glasg., Marine Lines, Bombay.

1878 *PHILIPSON, GEORGE HARE, M.D., M.A. Cantab., D.C.L. Durh., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., 10, Henrietta street, Cavendish square, W.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 13, South Eaton place, Eaton square. *Referee*, 1882-3. *Sci. Com.* 1870. *Lib. Com.* 1879-81.

1841 †PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. *Referee*, 1858, 1864-9, 1877-83. *Trans.* 4.

Elected

1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; Examiner in Forensic Medicine in the University of London; 30, Wimpole street. *Trans.* 1.

1846 POTTER, JEPHSON, M.D., F.L.S.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., *Secretary*, Physician to the Middlesex Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole street, Cavendish sq. S. (Oct.) 1883. *Referee*, 1879-83. *Trans.* 2.

1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Gt. Cumberland place, Hyde park. C. 1882-3. *Referee*, 1870-81. *Sci. Com.* 1870. *Lib. Com.* 1872-8.

1857 PRIESTLEY, WILLIAM OVEREND, M.D., Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

1883 PRINGLE, JOHN JAMES, M.B., C.M., 35, Bruton Street, Berkeley square.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1878 PYE, WALTER, Surgeon (with charge of out-patients) to St. Mary's Hospital; 4, Sackville street, Piccadilly.

1877 PYE-SMITH, PHILIP HENRY, M.D., Assistant-Physician to, and Lecturer on Physiology at, Guy's Hospital; Examiner in Physiology at the University of London; 54, Harley street, Cavendish square.

Elected

1850 †QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to University College Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Referee*, 1845-6, 1848, 1858-9. *Lib. Com.* 1846. *Trans.* 1. *Pro.* 2.

1852 †RADCLIFFE, CHARLES BLAND, M.D., *Treasurer*, Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8. V.P. 1879-80. T. 1881-3. *Referee*, 1862-6, 1870-8.

1871 RALFE, CHARLES HENRY, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square.

1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital, Nottingham.

1869 READ, THOMAS LAURENCE, 57, Gloucester road [11, Petersham terrace], Queen's gate, South Kensington.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Referee*, 1860-72, 1875-81. *Trans.* 1.

1882 REID, JAMES, M.D., Resident Physician to H.M. the Queen, Windsor Castle.

Elected

1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., *Vice-President*, Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1881 RICE, GEORGE, M.B., C.M. Edinb., The Infirmary, Plumstead, Kent.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., 13, Nelson crescent, Ramsgate.

1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.

1863 RINGER, SYDNEY, M.D., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80. *Trans.* 5.

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 22, Finsbury square. *Trans.* 2.

1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 23, St. John's street, Deansgate, Manchester.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Physician to University College Hospital; Physician to the Hospital for Consumption, Brompton; Examiner in Materia Medica in the University of London; 53, Harley street, Cavendish square, W.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity; Buxton, Derbyshire.

Elected

1843 RODEN, WILLIAM, M.D., Morningside, Kidderminster, Worcestershire.

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; Physician to the Royal Infirmary for Children and Women, Waterloo Bridge road [19, Ovington gardens, S.W.]. C. 1879-80.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. *Trans.* 1.

1882 ROUTH, AMAND J. McC., M.D., B.S., Physician to the Samaritan Free Hospital for Women; Assistant Physician Accoucheur to the Charing Cross Hospital; Obstetric Physician to the St. Marylebone General Dispensary; 6, Upper Montagu street, Montagu square.

1849 †ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1882 ROY, CHARLES L., M.D., The Brown Institution, Wandsworth road.

1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital; 22, New Hall street, Birmingham.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of Physiology in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., late Lecturer on Dental Surgery at Guy's Hospital; Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3. *Lib. Com.* 1862-3.

1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Waynflete Professor of Physiology in the University of Oxford; Museum, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

Elected

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1879 SANGSTER, ALFRED, B.A., M.B., Lecturer on Skin Diseases at the Charing Cross Hospital; 6, Savile row. *Trans.* 1.

1847 †SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Boreatton park, Baschurch, Shrewsbury.

1869 SANSOM, ARTHUR ERNEST, M.D., Physician (with charge of out-patients) to the London Hospital; 84, Harley street, Cavendish square. *Trans.* 2.

1845 †SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1879 SAVAGE, GEORGE HENRY, M.D., Bethlem Royal Hospital, St. George's road, Southwark.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., *Vice-President*, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 6.

1873 SCOTT, JOHN MOORE JOHNSTON, M.D., Lurgan, County Armagh.

1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street. *Trans.* 2.

1877 SEMON, FELIX, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 59, Welbeck street, Cavendish square. *Trans.* 1.

1875 SEMPLE, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington square. *Sci. Com.* 1879.

Elected

1873 *Shapter, Lewis, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1882 Sharkey, Seymour J., M.B., Assistant Physician to St. Thomas's Hospital; 77, Lambeth Palace road. *Trans.* 1

1840 Sharp, William, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1836 †Shaw, Alexander, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Referee*, 1842-3, 1846-50, 1855-7, 1865. *Lib. Com.* 1843. *Trans.* 4.

1859 Sibley, Septimus William, 7, Harley street, Cavendish square. C. 1882-3. *Sci. Com.* 1863. *Trans.* 4.

1848 †Sieveking, Edward Henry, M.D., *Librarian*, Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

1842 †Simon, John, C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1865 Sims, J. Marion, M.D., Surgeon to the New York State Women's Hospital; 267, Madison Avenue, New York.

1857 Siordet, James Lewis, M.B., Villa Preti, Mentone, Alpes Maritimes, France.

1882 Smith, Charles John, 54, Old Steyne, Brighton.

1879 Smith, E. Noble, Senior Surgeon and Surgeon to the Orthopædic Department of the Farringdon Dispensary; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 Smith, Eustace, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Hospital for Children, and to the City of London Hospital for Diseases of the Chest; 5, George street, Hanover square.

Elected

1872 SMITH, T. GILBART, M.A., M.D., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. *Trans.* 1.

1866 SMITH, HEYWOOD, M.A. M.D. Oxon., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 18, Harley street, Cavendish square.

1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-76. *Referee*, 1873-4, 1880-83. *Sci. Com.* 1867. *Trans.* 3.

1847 SMITH, WILLIAM J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1874 *SMITH, WILLIAM ROBERT, M.D., F.R.S. Ed., Physician to the Dispensary, Cheltenham; 15, Imperial square, Cheltenham.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 6, Harley street, Cavendish square. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans* 1.

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.

1875 SPITTA, EDMUND J., Ivy House, Clapham Common, Surrey.

1851 †SPITTA, ROBERT JOHN, M.D. Lond., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1882 STEAVENSON, WILLIAM EDWARD, M.B., 15, Henrietta street, Cavendish square.

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board; Greenford House, Sutton, Surrey.

Elected

1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

1879 *STIRLING, EDWARD CHARLES, late Assistant Surgeon and Lecturer on Physiology at St. George's Hospital; Adelaide, South Australia [care of T. Gemmell, Esq., 32, The Grove, Boltons, S.W.].

1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham,

1865 STOKES, WILLIAM, M.D., Examiner in Surgery, Queen's University, Ireland, and Surgeon to the Richmond Surgical Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1843 STORKS, ROBERT REEVE, Paris.

1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Professor of Clinical Ophthalmic Surgery in University College, and Senior Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5. *Lib. Com.* 1867-8.

1871 STRONG, HENRY JOHN, M.D., 64, North End, Croydon.

1863 STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. *Referee,* 1882-3.

1871 †SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1860 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.

1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health, Oldham; 244, Great Clowes street, Broughton, Manchester.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

Elected

1878 *SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles street, Birmingham. *Trans.* 4.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1875 TAY, WAREN, Surgeon to the London Hospital and Surgeon to the North Eastern Hospital for Children and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's street, Southwark. *Trans.* 1.

1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 9.

1862 THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Referee*, 1876-7. *Trans.* 1.

1857 THOMPSON, HENRY, M.D., Consulting Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

1852 †THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 7.

1862 THOMPSON, REGINALD EDWARD, M.D., *Vice-President*, Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

Elected

1881 THOMSON, WILLIAM SINCLAIR, M.D., 40, Ladbroke grove, Kensington park gardens.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman street, Portman square. *Trans.* 2.

1875 TIBBITS, HERBERT, F.R.C.P. Ed., 68, Wimpole street.

1848 †TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1880 TIVY, WILLIAM JAMES, 1, Tottenham place, Clifton, Bristol.

1872 TOMES, CHARLES S., B.A., F.R.S., Lecturer on Anatomy and Physiology at the Dental Hospital; 37, Cavendish square. *Lib. Com.* 1879.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

1882 TOOTH, HOWARD HENRY, M.B., 34, Harley street, Cavendish square.

1871 *TREND, THEOPHILUS W., M.D., Raeberry Lodge, Southampton.

1879 TREVES, FREDERICK, Assistant Surgeon to the London Hospital; 18, Gordon square. *Trans.* 1.

1881 *TREVES, WILLIAM KNIGHT, Surgeon to the Royal Sea Bathing Infirmary for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 TROTTER, JOHN WILLIAM, Surgeon-Major, Coldstream Guards; Bossall Vicarage, York.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 TUFNELL, THOMAS JOLLIFFE, Consulting Surgeon to the City of Dublin Hospital; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the London Hospital; 15, Finsbury square.

Elected

1873 TURNER, GEORGE BROWN, M.D., San Remo, Italy.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Joint Lecturer on Practical Surgery at St. George's Hospital; 49, Green street. Park lane.

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne gardens, Folkestone.

1876 VENN, ALBERT JOHN, M.D., Obstetric Physician to the Metropolitan Free Hospital; Physician to the Victoria Hospital for Children; 8, Upper Brook street, Grosvenor square.

1870 VENNING, EDGCOMBE, 87, Sloane street.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 141, Regent street.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to St. George's Hospital; 14, Park lane.

1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.

1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1867 *WALLIS, GEORGE, Surgeon to Addenbrooke's Hospital, Corpus Buildings, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth street, Portland place. *Lib. Com.* 1882-3. *Trans.* 3.

Elected

1852 †WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 41, Hyde park square. C. 1872. *Trans.* 1.

1851 †WALTON, HAYNES, Senior Surgeon to St. Mary's Hospital, 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1818 WARE, JOHN, Clifton Down, near Bristol.

1866 WARING, EDWARD JOHN, C.I.E., M.D., 49, Clifton gardens, Maida vale. *Referee*, 1881-3.

1877 WARNER, FRANCIS, M.D., Assistant Physician to the London Hospital and to the East London Hospital for Children; Lecturer on Botany at the London Hospital; 24, Harley street, Cavendish square. *Trans.* 1.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine, in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.

1879 WATERS, JOHN HENRY, M.D., C.M., 101, Jermyn street.

1878 WATNEY, HERBERT, M.D., 1, Wilton crescent, Belgrave square.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883. *Trans.* 1.

1879 WATTEVILLE, ARMAND DE, M.A., M.D., B.Sc., Medical Electrician to St. Mary's Hospital; 30, Welbeck street, Cavendish square.

Elected

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D., 82, Avenue des Termes, Paris.

1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. *Referee*, 1869-73, 1878-83. *Lib. Com.* 1864-73. *Trans.* 6.

1844 †WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

1878 WEISS, HUBERT FOVEAUX, 30A, George street, Hanover square.

1874 WELLS, HARRY, M.D., British Vice-Consulate, Gualeguay-chu, Entre Rios, Argentine Confederation.

1854 WELLS, SIR THOMAS SPENCER, Bart., Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 13. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; 29, Promenade des Anglais, Nice, Alpes Maritimes, France, and 2, Bolton row, Mayfair. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Medical Tutor and Medical Registrar at St. Bartholomew's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Assistant Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. *Trans.* 2.

1882 WHARRY, CHARLES JOHN, M.D., Resident Superintendent, Government Civil Hospital, Hong Kong.

1881 WHARRY, ROBERT, M.D., 6, Gordon square.

Elected.

1878 WHARTON, HENRY THORNTON, M.A., Surgeon to the Kilburn Dispensary; 39, St. George's road, Kilburn.

1828 WHATLEY, JOHN, M.D.

1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square.

1849 WHITE, JOHN.

1881 WHITE, WILLIAM HALE, M.D., Demonstrator of Anatomy at Guy's Hospital; 4, St. Thomas's street, Southwark.

1881 *WHITEHEAD, WALTER, F.R.S. Ed., Surgeon to the Manchester Royal Infirmary; 24, St. Ann's square, Manchester.

1877 WHITMORE, WILLIAM TICKLE, 7, Arlington street, Piccadilly.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1870 *WILKIN, JOHN F., M.D. and M.C., New Beckenham, Kent.

1883 *WILKINSON, THOMAS MARSHALL, Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; Silver Street, Lincoln.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

1865 †WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish sq. C. 1880-81. *Referee*, 1882-3. *Trans.* 2.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]. C. 1849-50. V.P. 1860-1. P. 1873-4. *Referee*, 1843-4. *Sci. Com.* 1862. *Trans.* 1.

Elected

1859 *WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 47, Upper Brook street, Grosvenor square. *Lib. Com.* 1880-3. *Trans.* 3.

1881 WILLIAMS, DAWSON, M.B., 4, Oxford and Cambridge Mansions, Marylebone road.

1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 11, Queen Anne street, Cavendish square. *Referee*, 1878-83. *Lib. Com.* 1876-82.

1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy 19, Whitehall place.

1839 †WILSON, SIR ERASMUS, Bart., F.R.S., late Professor of Dermatology, Royal College of Surgeons of England Bungalow, Westgate-on-Sea, and 11, Manchester square. C. 1877. *Lib. Com.* 1845. *Trans.* 2.

1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 WISE, THOMAS ALEXANDER, M.D., Inchrye Abbey, Newborough, Fife, N.B.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 57, Harley street, Cavendish square.

1851 †WOOD, JOHN, F.R.S., Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; Examiner in Surgery in the University of London; 61, Wimpole street, Cavendish square. C. 1867-8. V.P. 1877-8. *Referee*, 1871-6, 1880-83. *Lib. Com.* 1866. *Trans.* 3.

1848 †WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.

Elected

1883 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., Assistant Medical Officer, Bethlem Royal Hospital.

1881 WOODMAN, SAMUEL, Consulting Surgeon to the Ramsgate and St. Lawrence Royal Dispensary; 5, Prospect terrace, Ramsgate.

1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1842 WORTHINGTON, WILLIAM COLLINS, London road, Lowestoft, Suffolk. *Trans.* 3.

1865 WOTTON, HENRY, M.D., 15, Notting hill terrace.

1878 YEO, GERALD F., M.D., M.Ch., Professor of Physiology in King's College, London; King's College, Strand.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1883 CARPENTER, WILLIAM BENJAMIN, M.D., C.B., LL.D., F.R.S., Corresponding Member of the Institute.

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.

1883 FRANKLAND, EDWARD, M.D., D.C L., Ph.D., F.R.S., Professor of Chemistry in the Royal School of Mines; Corresponding Member of the French Institute; Royal College of Chemistry, South Kensington Museum, and the Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, M.D., C.B., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.

1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

1878 LUBBOCK, SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Bromley, Kent.

Elected

1847 OWEN, RICHARD, C.B., D.C.L., LL.D., F.R.S., Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, Mortlake.

1883 PARKER, WILLIAM KITCHEN, F.R.S., Hunterian Professor of Comparative Anatomy in the Royal College of Surgeons; 36, Claverton street, Pimlico.

1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., Lucasian Professor of Mathematics in the University of Cambridge; Secretary to the Royal Society, &c.; Lensfield Cottage, Cambridge.

1883 THOMSON, ALLEN, M.D., D.C.L., LL.D., F.R.S., 66, Palace gardens terrace, W.

1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1878 BACCELLI, GUIDO, M.D., Professor of Medicine at Rome.

1883 BIGELOW, HENRY J., M.D., Professor of Surgery at Harvard University, and Surgeon to the Massachusetts General Hospital.

1876 BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna; Vienna.

1883 CHARCOT, J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor at the Faculty of Medicine of Paris; Member of the Academy of Medicine; Quai Malaquais 17, Paris.

1864 DONDERS, FRANZ CORNELIUS, M.D., LL.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1883 DUBOIS REYMOND, EMIL, M.D., Professor in Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

1876 EDWARDS, H. MILNE, M.D., Member of the Institute of France, and of the Academy of Medicine; Dean of the Faculty of Sciences and Professor at the Museum of Natural History of Paris; 57, Rue Cuvier, Paris.

1835 EKSTRÖMER, CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden; President of the College of Health, and Director-General of Hospitals; Stockholm.

Elected

1868 GROSS, SAMUEL D., M.D., F.C.P. Philad., D.C.L. Oxon., LL.D. Camb., Emeritus Professor of Surgery in the Jefferson Medical College of Philadelphia.

1878 GUENEAU DE MUSSY, NOEL, M.D., Member of the Academy of Medicine; Physician to the Hôtel Dieu; 4, Rue St. Arnaud, Paris.

1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.

1873 HELMHOLTZ, HERMANN LUDWIG FERDINAND, Professor of Physics and Physiological Optics; Berlin.

1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.

1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

1868 KÖLLIKER, ALBERT, Professor of Anatomy in the University of Würzburg.

1856 LANGENBECK, BERNHARD, M.D., late Professor of Surgery in the University of Berlin.

1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1883 PASTEUR, LOUIS, Member of the Institute of France (Academy of Sciences).

1878 SCANZONI, FRIEDREICH WILHELM VON, Royal Bavarian Privy Councillor, and Professor of Medicine in the University of Würzburg.

1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1819 Jas. M. Arnott, F.R.S.
1828 Cæsar H. Hawkins, F.R.S.
1833 Sir George Burrows, Bt., M.D., F.R.S.
Thomas A. Barker, M.D.
1835 Richard Quain, F.R.S.
Thomas A. Nelson, M.D.
1836 Alexander Shaw.
J. George French.
1837 Thomas Blizard Curling, F.R.S.
George Busk, F.R.S.
1838 Charles Hawkins.
Henry Spencer Smith.
1839 T. Graham Balfour, M.D., F.R.S.
Sir W. J. Erasmus Wilson, Bt., F.R.S.
Fred. Le Gros Clark, F.R.S.
James Dixon.
1840 Chas. J. B. Williams, M.D., F.R.S.
Charles Hutton, M.D.
Samuel A. Lane.
Sir James Paget, Bt., F.R.S.
1841 Sir Henry A. Pitman, M.D.
William Bowman, F.R.S.
John Parkin, M.D.
Paul Jackson.
1842 Charles West, M.D.
Frederic Weber, M.D.
John Simon, C.B., F.R.S.
John Erichsen, F.R.S.
Sir Oscar M. P. Clayton.
1843 Robert Greenhalgh, M.D.
Sir Prescott G. Hewett, Bt., F.R.S.
Henry Lee.
Wm. White Cooper.
1843 Luther Holden.
Edward Newton.
1844 Arthur Farre, M.D., F.R.S.
William Wegg, M.D.
Thomas King Chambers, M.D.
Edwin Humby.
1845 Samuel Cartwright.
George D. Pollock.
Thomas Taylor.
Sir Edwin Saunders.
William Oliver Chalk.
Edward U. Berry.
Benjamin Ridge, M.D.
1846 John A. Bostock.
Barnard Wight Holt.
Carsten Holthouse.
1847 W. H. O. Sankey, M.D.
George Johnson, M.D., F.R.S.
1848 Edward H Sieveking, M.D.
Edward Ballard, M.D.
William Wood, M.D.
Thomas Hawksley, M.D.
Edward John Tilt, M.D.
John Clarke, M.D.
John Gay.
John Gregory Forbes.
1849 Hugh J. Sanderson, M.D.
C. H. F. Routh, M.D.
Edmund L. Birkett, M.D.
George T. Fincham, M.D.
Sir William W. Gull, Bt., M.D., F.R.S.
1850 Richard Quain, M.D., F.R.S.
George Roper, M.D.
1851 Sir Wm Jenner, Bt., M.D., F.R.S.

1851 H. Haynes Walton.
John Birkett.
John A. Kingdon.
Peter Y. Gowlland.
John Marshall, F.R.S.
John Wood, F.R.S.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
George Gaskoin.
1852 C. Bland Radcliffe, M.D.
Walter H. Walshe, M.D.
William Adams.
John Cooper Forster.
Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Alfred Baring Garrod, M.D., F.R.S.
Samuel O. Habershon, M.D.
Sir Thomas Spencer Wells, Bt.
1855 W. M. Graily Hewitt, M.D.
J. Burdon Sanderson, M.D., F.R.S.
J. Russell Reynolds, M.D., F.R.S.
Walter John Bryant, M.D.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
Henry Thompson, M.D.
Hermann Weber, M.D.
George Owen Rees, M.D., F.R.S.
John Whitaker Hulke, F.R.S.
John Morgan.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 Fred. George Reed, M.D.
William Chapman Begley, M.D.
John William Ogle, M.D.
Wilson Fox, M.D., F.R.S.
John Fremlyn Streatfeild.
Francis Harris, M.D.
1859 Wm. Howship Dickinson, M.D.
William Scovell Savory, F.R.S.
Edwin Thomas Truman.
Francis Hird.
Richard Barwell.
Edward Tegart.
Septimus William Sibley.
William E. Stewart.
1860 Sir Andrew Clark, Bt., M.D.
John Maclean, M.D.
Sigismund Sutro, M.D.
William Ogle, M.D.
1860 Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 Robert Barnes, M.D.
William Spencer Watson.
William Henry Holman.
1862 James Andrew, M.D.
Lionel Smith Beale, M.B., F.R.S.
Thomas H. Tuke, M.D.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
William Henry Brace, M.D.
George Cowell.
M. Berkeley Hill.
1863 Octavius Sturges, M.D.
John Langdon H. Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D.
Thomas Smith.
Arthur B. R. Myers.
Arthur E. Durham.
William Sedgwick.
1864 Charles Hilton Fagge, M.D.
George Buchanan, M.D., F.R.S.
Charles Derby Waite, M.B.
John Harley, M.D.
Walter John Coulson.
Thomas William Nunn.
Francis Mason.
Jos. Gillman Barratt, M.D.
1865 Charles Robert Drysdale, M.D.
James Edward Pollock, M.D.
William Cholmeley, M.D.
Reginald Southey, M.D.
George Fielding Blandford, M.D.
Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
William Morrant Baker.
John Langton.
Frederick James Gant.
Alfred Willett.
Bowater John Vernon.
Alfred Cooper.
Christopher Heath.
Henry Wotton.
1866 Thomas Fitzpatrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
John Crockett Fish, M.D.
William Selby Church, M.D.
Edward John Waring, M.D.

1866 William Fairlie Clarke, M.D.
Philip H. Harper.
1867 William Henry Day, M.D.
Achille Vintras, M.D.
Richard Douglas Powell, M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac.
Thomas Pickering Pick.
John Astley Bloxam.
Charles Arthur Aikin.
Samuel Hill, M.D.
Colomiati Meredyth, M.D.
1868 H. Charlton Bastian, M.D., F.R.S.
William Henry Broadbent, M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
John Cockle, M.D.
Thomas Crawford, M.D.
T. Henry Green, M.D.
William Rhys Williams, M.D.
Walter Moxon, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
William Henry Freeman.
1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
John Wickham Legg, M.D.
Charles Elam, M.D.
Thomas Laurence Read.
1870 Alfred Meadows, M.D.
William Wadham, M.D.
J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
1871 William Cayley, M.D.
Charles Henry Ralfe, M.D.
Arthur Julius Pollock, M.D.
Thomas L. Brunton, M.D., F.R.S.
Henry Gawen Sutton, M.D.
J. Hughlings Jackson, M.D., F.R.S.
Henry Sutherland, M.D.
George Vivian Poore, M.D.
Walter Rivington.
Marcus Beck.
Edward Bellamy.
William F. Butt.
Benjamin Duke.
1872 Gilbart Smith, M.D.
Thomas B. Christie, M.D.
George B. Brodie, M.D.
John Williams, M.D.
1872 Sir J. Fayrer, M.D., F.R.S.
Charles S. Tomes, B.A., F.R.S.
William Bartlett Dalby.
1873 William Miller Ord, M.D.
Frederick Taylor, M.D.
Norman Moore, M.D.
John Curnow, M.D.
William R. Gowers, M.D.
William Guyer Hunter, M.D
Charles Creighton, M.D.
Jeremiah McCarthy.
Wm. Johnson Smith.
Robert William Parker.
Alex. O. McKellar.
Henry T. Butlin.
Charles Higgens.
William J. Walsham.
Edward Milner.
1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
Alfred B. Duffin, M.D.
James H. Aveling, M.D.
Evan B. Baxter, M.D.
John M. Bruce, M.D.
Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Henry G. Howse.
Herbert William Page.
Frederic Durham.
John J. Merriman.
1875 Thomas T. Whipham, M.B.
Francis Charlewood Turner, M.D.
William K. Murphy, M.D.
Herbert Tibbits, F.R.C.P. Ed.
Robert Hunter Semple, M.D.
Thomas Crawford Hayes, M.D.
Charles Henry Carter, M.D
Fletcher Beach, M.B.
Samuel Osborn.
Waren Tay.
Edmund J. Spitta.
1876 Thomas Barlow, M.D.
John C. Bucknill, M.D., F.R.S.
Wm. Lewis Dudley, M.D.
Albert J. Venn, M.D.
John Knowsley Thornton.
Charles Macnamara.
John N. C. Davies-Colley.
1877 Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.B.
Alfred Pearce Gould.

1877 J. Rickman Godlee.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.B.
John Abercrombie, M.B.
J. Matthews Duncan, M.D., F.R.S. Ed.
Henry de Fonmartin, M.D.
George Allan Heron, M.D.
Joseph A. Ormerod, M.B.
P. Henry Pye-Smith, M.D.
Edward Nettleship.
William Henry Bennett.
Benj. T. Lowne.
William T. Whitmore.
1878 Jas. Crichton Browne, M.D.
Fred. T. Roberts, M.D.
Joseph Lister, F.R.S.
Clinton T. Dent.
John H. Morgan.
Walter Pye.
Gerald F. Yeo, M.D.
Donald W. Charles Hood, M.B.
Henry Gervis, M.D.
Herbert Watney, M.D.
Richard Davy.
Hubert Foveaux Weiss.
Henry Thornton Wharton.
1879 Alfred Sangster, M.B.
Edward Woakes, M.D.
Armand de Watteville, M.D.
Malcolm A. Morris.
A. E. Cumberbatch.
Edmund Owen.
Arthur E. J. Barker.
Frederick Treves.
Horatio Donkin, M.B.
Thomas John Maclagan, M.D.
David White Finlay, M.D.
Andrew Clark.
S. Hamilton Cartwright.
John H. Waters, M.D.
Francis Henry Champneys, M.D.
William Watson Cheyne.
William Munk, M.D.
George Henry Savage, M.D.
H. H. Clutton, M.A.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.B
F. G. Dawtrey Drewitt, M.D.
1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Vincent Dormer Harris, M.D.
1880 Edmund D. Maddick.
Jas. John MacWhirter Dunbar, M.B.
James William Browne, M.B.
William Appleton Meredith, M.B.
Alexander Hughes Bennett, M.D.
Malcolm Macdonald McHardy.
A. Boyce Barrow.
William Murrell, M.D.
Bernard O'Connor, A.B., M.D.
Leslie Ogilvie, M.B.
George Lockwood Laycock, M.B.
George Ogilvie, M.B.
Charles Edward Beevor, M.D.
Thomas Colcott Fox, M.B.
George Henry Makins.
1881 Francis de Havilland Hall, M.D.
Robert Wharry, M.D.
Cecil Yates Biss, M.B.
Richard Clement Lucas.
Stephen Mackenzie, M.D.
James Anderson, M.D.
William Hale White, M.D.
Eustace Smith, M.D.
William Sinclair Thomson, M.D.
Percy Kidd, M.D.
Oswald A. Browne, M.A.
Audley Cecil Buller.
W. Bruce Clarke, M.B.
Dawson Williams, M.B.
George Lindsay Johnson, M.A.
Henry Edward Juler.
Jonathan F. C. H. Macready.
Henry Howard, M.B.
Samuel Nall, M.B.
C. B. Lockwood.
1882 Philip J. Hensley, M.D.
Ernest Clarke.
Isidore I. Lyons.
John Barclay Scriven.
Yelverton Dawson, M.D.
Charles L. Roy, M.D.
George Robertson Turner.
David Alexander King, M.B.
Howard Henry Tooth, M.B.
Herbert Isambard Owen, M.D.
Charles R. B. Keetley.
Joseph Mills.
A. T. Myers, M.D.
Anthony A. Bowlby.
Amand J. McC. Routh, M.D.
Seymour J. Sharkey, M.B.
William Lang.
Henry Radcliffe Crocker, M.D.
William Edward Steavenson, M.B.

1882 D. Astley Gresswell, M.B.
1883 Edwin Clifford Beale, M.A., M.B.
James Kingston Fowler, M.B.
James Frederic Goodhart, M.D.
John Charles Galton, M.A.
Walter Hamilton Acland Jacobson.
Edward Joshua Edwardes, M.D.
Walter H. H. Jessop, M.B.
Walter Edmunds, M.D.

1883 William E. Ramsden Wood, M.D.
Victor A. Horsley.
Dudley Wilmot Buxton, M.B.
Charles Douglas F. Phillips, M.D.
Hutchinson Royes Bell.
Angel Money, M.D.
John James Pringle, M.B.
Henry Roxburgh Fuller, M.B.
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ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

Regulations relative to the publication of the 'Proceedings of the Society.'

That, as a general rule, the 'Proceedings' will be issued every two months, subject to variations dependent on the extent of matter to be printed.

That a Copy of the 'Proceedings' will be sent, postage free, to every Fellow of the Society resident in the United Kingdom.

That the 'Proceedings of the Society' may be obtained by non-members at the Society's House, 53, Berners Street, on pre-payment of an annual subscription of five shillings, which may be transmitted either by post-office order or in postage-stamps; —this will include the expense of conveyance by post to any part of the United Kingdom; to other places they will be sent, carriage free, through a bookseller, or by post, the receiver paying the foreign charges.

That a notice of every paper will appear in the 'Proceedings.' Authors will be at liberty, on sending their communications, to intimate to the Secretary whether they wish them to appear in the 'Proceedings' *only*, or in the 'Proceedings' and 'Transactions;' and in all cases they will be expected to furnish an Abstract of the communication.

The Abstracts of the papers read will be furnished to the Journals as heretofore.

ADDRESS

OF

JOHN MARSHALL, F.R.S.,

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1st, 1883.

Gentlemen, Fellows of the Royal Medical and Chirurgical Society,—In addressing myself to the customary duty of referring to the lives and work of those whom the Society has lost by death during the past year, I find that these losses have been so numerous and important that I must limit myself exclusively to that task, deferring to a future opportunity the devotion of a part of my address to other subjects, such as the general progress of Medicine and Surgery, or to questions relating to this Society.

The deceased *non-resident* Fellows, nine in number, include three (Drs. Hood, Hogg, and Morehead) who served with the army in India, three others (Drs. Elliot, Cross, and McEwen) who had been engaged in practice in large provincial towns, and three (Drs. Johnston, Williams, and Budd, who had retired from metropolitan practice to reside in the country. Another deceased physician, Sir James Alderson, though he had for some time past left the Society, was connected with it for many years, partly as a non-resident but chiefly as a resident Fellow.

There are eight deceased *resident* Fellows :—Drs. Lyell and Silver, Mr. Clover, Dr. Taylor and Mr. Critchett, Dr. Peacock and Sir Thomas Watson. Besides these, two Honorary Fellows have died since our last anniversary—Dr. Draper, of New York, and Charles Darwin.

If I speak of the individual members of each of these

classes in the order of the duration of their Fellowship, commencing with the youngest, this order will be found to correspond nearly always with their respective ages, influence, and position in our profession. In such portions of my remarks as are not based upon my acquaintance with the deceased, or on circumstances regarding them which have come within my own cognisance, I have been much indebted to obituaries which have already appeared in the public journals.

Francis Edward Charles Hood, the first and the youngest on our obituary list, died at Agra, of remittent fever, on the 15th of February, 1882, and therefore before our last annual meeting. He had been a Fellow of our Society only three years. The son of a late Fellow, Sir William C. Hood, who was formerly a physician to Bethlehem Hospital, and the younger brother of Dr. Donald W. C. Hood, who is now a Fellow, Surgeon Francis Hood served to illustrate that family inclination towards the pursuit of Medicine, which is so frequently noticed, and of which other instances will presently be referred to.

Educated at Guy's Hospital, and having obtained his qualifications in 1875, he joined the Army Medical Department five years afterwards, and went to India in the autumn of 1880. Falling a victim to climatic disease at the early age of twenty-eight, he had no time to reap such honours as would most probably have awaited him had his life been spared.

Francis Roberts Hogg, who died at Morar, Gwalior, on September 27th, 1882, at the age of forty-six, had been a Fellow of the Society for ten years. Having obtained his qualifications in 1857-8, and passed through the Army Medical School at Netley, he immediately entered for service with the Royal Artillery, proceeded to India, and rose to the rank of Surgeon-major (1873). At home, Dr. Hogg was employed for more than ten years as Surgeon to the Woolwich Hospital for Soldier's Wives and Families. Upon his Indian experience, he founded an interesting little

work, on 'Marriage in the Army Medically Considered' (1873), also his 'Practical Remarks on the Health of European Families in India,' and his 'Indian Notes' (1880). The period of his appointment at Woolwich was exceptionally prolonged, owing to the high esteem in which he was there held; and his comparatively early death, very soon after his going again to India, was deeply regretted by all persons, of whatever grade, who had been associated with him, or had been under his professional care.

Charles Morehead, a distinguished Indian Medical Officer, who died on the 24th of last August, at the advanced age of seventy-four, whilst on a visit at Wilton Castle, near Redcar, in Yorkshire, was for twenty-one years a Fellow of this Society, and for a short time was appointed a Referee.

The son of the Rev. R. Morehead, of Edinburgh, afterwards Rector of Essington, he was born in the Scotch Capital (1807), and received his early medical training, and his first professional rank (M.D.) in that city, where he had been a clinical clerk and a favourite pupil of Dr. Alison. He appears also to have studied in Paris under Louis, with whom he long continued to correspond.

At the early age of twenty-two (1829)—for service began soon in those days—he joined the Bombay Medical Department; but, promotion being slow, he was not gazetted Surgeon-major until 1860. His ability was, however, soon recognised, for he served for three years on the staff of Governor Gray, of Bombay, and in 1838 was appointed Surgeon to the European and Native General Hospital in that city. In 1840 he was chosen, for his special qualifications, secretary to the Board of Native Education, an office which he held for five years, when he was appointed (1845) the first Principal, and also the first Professor of Medicine, in the Grant Medical College of Bombay for Natives of Western India. He likewise became Surgeon to the Jamsetjee Jeejeebhoy Hospital, and was, for a time, President of the Bombay Medical and Physical Society.

In evidence of his intellectual activity, and of his love of clinical observation, he wrote, besides several slight papers, an important work in two volumes, entitled 'Clinical Researches on Diseases in India,' a second edition of which, considerably modified in its opinions, appeared in 1860.

After thirty years of continuous and arduous employment in India, Dr. Morehead returned home on furlough (1859), became F.R.C.S. Ed. (1860), F.R.S. Ed., Surgeon-major in the army, and Deputy Inspector-general of Hospitals, and finally retired from active service in 1862. As special marks of distinction he was appointed Honorary Surgeon to the Queen (1861), and more recently (1881) a Companion of the Order of the Indian Empire.

Actuated by the teachings of Alison and Louis, he was an exact observer and a careful note-taker, and accordingly he excelled in diagnosis, and was a reliable instructor. He achieved a great reputation in India, where his memory will long survive as a worthy representative of our profession, not only as an accomplished physician and surgeon, but as a most efficient public servant.

Richard Cross, of Scarborough, the first of the three provincial brethren whom we have lost during the past year, had been a non-resident Fellow of the Society for twenty-two years. He entered the profession as a Surgeon and Apothecary in 1839-40, became M.D. of St. Andrew's in 1852, and only a short time since (1881) was made a Fellow of the Royal College of Surgeons of England. He died on the 18th of last November, at the age of sixty-four, about twelve hours after he had submitted to an amputation above the knee, for a tedious and painful disease of that joint.

Dr. Cross was an admirable example of a large class of provincial practitioners, who, by their ability and skill, acquire a widely spread local reputation in their profession, and by their energy and upright character gain a correspondingly distinctive social position. Eminent in

both private and public practice, he so far secured the suffrages of his fellow townsmen as to be chosen Mayor. The great respect in which he was held, and the sympathy excited by the special circumstances of his death, were touchingly shown by the civic and popular demonstration which took place at his funeral.

Robert Elliot, of Carlisle, who died on the last day of 1882, at the age of seventy-one, was for upwards of twenty-one years a non-resident Fellow. Educated at Edinburgh, where he took his degree in 1836, he studied afterwards in Paris and at Heidelberg. Commencing practice at Gateshead and Newcastle, at the age of twenty-eight, he was soon after appointed Lecturer on Materia Medica and Hygiene in the Newcastle Medical School, speedily proving himself to be a ready and forcible speaker, and an excellent teacher.

In 1848, when he was thirty-five years of age, he was summoned to Carlisle to take part in the extensive practice established by his family in that city; the Elliots furnishing another instance of a strong proclivity to Medicine, exhibiting itself in three generations.

At Carlisle, as was natural to a teacher of Hygiene, Dr. Robert Elliot threw himself into the vortex of questions and work connected with the sanitary improvement of his native city, and with the social advancement of its citizens. He advocated and supported the public library and reading room; he lectured on ventilation, water-supply, and sewerage, some of his papers on these subjects appearing in the 'Transactions of the Social Science Congress.' His influence and popularity were testified by his election to the mayoralty of Carlisle in 1855, and by his subsequent appointment to the offices of Justice of the Peace, of Coroner for the city (1873), and of first Medical Officer of Health (1874), which two last-named positions he held until his death.

An Edinburgh graduate of 1836, Dr. Elliot became a F.R.C.P.L. in 1873. He was Physician and afterwards consulting Physician to the Free Dispensary, to the

Hospital for Chronic Diseases, and to the Fever Hospital, in Carlisle. He was at one time an Examiner in Medicine for the Durham University, and was also a President of the Northern branch of the British Medical Association. He contributed several interesting papers to medical journals. He has been described as "one of the oldest, most energetic, and most respected physicians in the North of England;" and he died full of years and honours.

William McEwen, of Chester, who died of paralysis on the 1st of August last, as it would appear at about the age of seventy, had been a non-resident Fellow of this Society for as many as thirty-six years. An Edinburgh licentiate of 1833, he became a St. Andrew's M.D. in 1845, a Member of the Royal College of Physicians of London in 1859, and a Fellow of the Royal College of Surgeons of Edinburgh in 1871. He contributed two papers to the 'British Medical Journal,' and an account of a case of hydatids to Churchill's 'Diseases of Women.' He held, in succession, the offices of house Surgeon to the Chester Infirmary, and of resident Surgeon, Superintendent, and ultimately, visiting Physician to the Cheshire County Lunatic Asylum. At the time of his death he was Surgeon to Her Majesty's Prison in Chester Castle. In token of the general estimation in which he was held, he had been made a Justice of the Peace for the City of Chester.

Of a most genial nature, as I have myself experienced, and of keen and quick perception, Dr. McEwen was an active and sound practitioner, and he was highly valued and thoroughly trusted in his social as well as in his professional relations. Whether individual physicians or surgeons, and whether the profession at large gain or lose by becoming engaged in civic work, are questions which can only be decided on the merits of individual examples; but the instances which we have here before us, in the cases of Dr. Cross, of Scarborough, Dr. Elliot, of Carlisle, and Dr. McEwen, of Chester, prove that when experienced members

of our profession choose to devote themselves to the discharge of public duty, they are welcomed and honoured by their fellow citizens, and may in turn be said to reflect honour on their own calling.

William Woods Johnston, who, until recently, resided in London, died on the 2nd of last month, at Tunbridge Wells, at the age of eighty-three. He was an Edinburgh M.D., and a Member of the College of Physicians of London. He formerly lived in Java and was in possession of considerable wealth.

I have no further particulars of his career. He has been a Fellow of this Society for about fifteen years; he was very secluded, and, before leaving London, made much use of the Society's library.

Joseph Williams, formerly of Tavistock Square, but lately of Holmhurst, Twickenham, is the second of the three deceased Fellows who have retired from London to seek rest in a country life. As a resident and a non-resident Fellow, he held a place in our ranks for twenty-three years. He died at Holmhurst, on the 20th of last March, at the age of sixty-seven.

Educated at Guy's Hospital Medical School, at Trinity College, Dublin, in Edinburgh, and in Paris, he joined the College of Surgeons of England in 1836, became M.D. of Edinburgh in 1839, and, much later in life (1859) a Member of the Royal College of Physicians of London. His earliest literary effort was a systematic work, on 'The Anatomy and Pathology of the Ear' (1840); but he afterwards abandoned the subject of Otology and turned his attention to cerebral disease, gaining the Lord Chancellor Sugden's prize, given at Trinity College, Dublin, for an essay on "Narcotics and other remedial agents calculated to produce Sleep in Insanity (1845). He afterwards published, besides smaller papers, an important work on 'Insanity,' which reached a second edition (1852).

Whilst resident in London, and engaged in practice, Dr. Joseph Williams was held in much respect and gave

importance to the offices of Physician to the Home for Gentlewomen, and Physician to the St. Pancras Female School, which were entrusted to him. I myself well remember him as a most amiable and enthusiastic colleague on the Committee for Establishing the Public Baths and Wash-houses in the Hampstead Road, the first which were opened in this part of London, and the second in the entire Metropolis. It is now some years since, that, finding his highly nervous temperament unsuited to withstand the wear and tear of an anxious branch of practice, he resolved to prolong his days, and enhance the pleasure of existtence, by retiring to his final home and resting-place near Twickenham.

George Budd, who died on the 14th of last March (1882), at his country residence in Ashleigh, near Barnstaple, recalls to mind probably the most remarkable example of the devotion of numerous members of a large family to the study and practice of Medicine. Happily, too, they have been blest with lives sufficiently prolonged to enable them to reach good rank in their respective positions in our profession. From the home of a busy, trusted, and respected practitioner in the village of North Tawton, in Devonshire, there proceeded out of a family of nine sons and one daughter, seven sons who chose a medical career. Of these seven, five were educated at Cambridge, and became Wranglers. The third in this gifted fraternity, George, was entered at Caius College, and came out third Wrangler in 1831, in the same year with Dr. George Paget, the present Regius Professor of Physic in that University, who was eighth on the Wrangler's list. Awarded a Fellowship in his College, George Budd afterwards pursued his medical studies in Paris, and subsequently at the Middlesex Hospital. He was, very early in his career, attached to the Dreadnought Seamen's Hospital Ship at Greenwich, with which he was finally connected as Consulting Physician. So far back as 1836, he was made a Fellow of the Royal Society. Elected a Fellow of this Society in 1839, he remained associated with it for twenty-eight years as a

resident Fellow, and was for fifteen years a non-resident Fellow. During the former period he served on the Library Committee, as a Referee, as a member of Council, and finally, as a Vice-President (1857). In 1840, having graduated M.D. at Cambridge, Dr. George Budd was elected Professor of Medicine at King's College, on the resignation of Dr., afterwards Sir Thomas Watson, who survived his successor and junior by several months. In the same year he was appointed, with the late Dr. Todd, Physician to the newly-built King's College Hospital. In 1841, he became a Fellow of the Royal College of Physicians, and in due time he was elected Censor (1845—47), and placed on the Council (1862). In 1863, after twenty-three years of valuable service at King's College and its Hospital, he retired from those institutions, with the title of Honorary Fellow of the College. A few years later (1867), warned by the approach of failing health, he retired from London, and from a large practice, which he had conducted for nearly a quarter of a century. He then visited the Continent, wintered in Italy, and, on returning to England in 1869, went to reside near his brother, Dr. Richard Budd, of Barnstaple, and participated actively, for many years, in the enjoyment of country pursuits, including even hunting. Although thus withdrawn from professional life, he was not overlooked by his old University; for, much to his delight, he was, only three years since, and on the same occasion as Sir George Burrows, made an Honorary Fellow of Caius College, the scene of his first intellectual efforts. Latterly, however, his strength gradually failed, and he died on the 14th March, 1882, after a brief illness, just a few months after the completion of his seventy-fourth year.

Dr. George Budd's early mathematical triumph at Cambridge was followed by such zealous labour in the study of Medicine, that when appointed to the Seamen's Hospital Ship at Greenwich, he was already noted as an acute clinical and pathological observer, and was enabled, from the hospital records, to contribute two valuable papers on

"Cholera," to our 'Transactions," as well as three others on different subjects.

It was on board the Dreadnought likewise, that he collected the chief data for his standard work on 'Diseases of the Liver,' which, between the years 1845 and 1857, passed through three editions. He also wrote a treatise on "Organic Diseases and Functional Disorders of the Stomach," and articles on "Cholera," and on "Scurvy," in Tweedie's 'Library of Medicine.' In addition to the reputation dependent on these important literary labours, his lectures on the "Principles and Practice of Medicine," given for so many years at King's College, gained him great distinction, amply fulfilling, as Sir Thomas Watson declared in 1871, the prophecy which that still greater teacher had made concerning him on his appointment in 1840. His important services at King's College secured to him the respect and gratitude of his colleagues, contemporaries, and pupils, from the last of whom he carried with him, on his retirement to his native county, a substantial testimonial.

Some of us, at least, can remember his tall and spare frame, his academic style, his keen glance, and intellectual physiognomony. As a lecturer, he was eloquent and learned; as a bedside teacher, earnest, emphatic, and practical. In his social relations he was much esteemed. He was one of the most distinguished members of a remarkable medical family, and, together with his brother Dr. Wm. Budd, will leave durable traces in the history of British Medicine, and a name of which his little Devonshire birthplace may long continue to be proud.

Sir James Alderson, who died on the 13th of September last, at the great age of eighty-seven, had ceased for many years to be a Fellow of this Society, and, perhaps, in strict accordance with custom, his name should not be mentioned in this obituary record. But he was associated with the Society for forty-six years (1826 to 1872), first as a resident Fellow for three years, then as a non-

resident Fellow for sixteen years (whilst he was practising in his native town, Hull), and then again, on his return to London, as a resident Fellow for nearly twenty-seven additional years. He was Secretary (1829), on the Council (1848), Treasurer (1849), Vice-President (1852-3) and President (1855-6). Moreover, three contributions from his hand appear in our 'Transactions,' viz. "On the Pathology of Whooping Cough," vol. xvi; "A Notice of the Effects of Lead upon the System," vol. xxii; and "A Case of Skin Disease, with Partial Hypertrophy of the Mammary Gland," vol. xxxvii. It would, therefore, seem unjust to omit the name of one who had done such service to the Society, and discourteous to his memory to shut out from review the chief incidents in his career.

The son of an able physician in Hull, and the brother of Baron Alderson, the well-known judge, James Alderson, who was born in the last century, entered at Pembroke College, Cambridge, came out as sixth Wrangler in 1822, and was made Fellow of his College. From 1826 to 1829 he was in London, and then he took his M.D. degree at Oxford. He next practised at Hull, whence (1845) he once more returned to metropolitan work. He was a Fellow of the Royal Society. For many years associated with the Royal College of Physicians of London, as Fellow (1830), Censor, and Councillor, he was elected President of the College in 1867, and retained that office until 1870. It was during his presidency that he received the honour of Knighthood; he also became Physician Extraordinary to the Queen, and was made a D.C.L. of the University of Oxford.

For about twenty years Sir James Alderson was Senior Physician to St. Mary's Hospital, to which institution he had been attached from its commencement. Besides his contributions to our 'Transactions,' he published a very early paper, "On the Motion of the Heart" (1825); "A History of the Cholera at Hull" (1832); "Observations on Diseases of the Stomach and Alimentary Canal" (1847); "The Lumleian Lectures on the Effects

of Lead" (1853); a paper "On Acute Rheumatism;" a pamphlet on 'Medical Reform;' the 'Harveian Oration' (1867); and 'Some Observations on Sea Sickness' (1872).

Having retired from all professional engagements, and almost from professional society, for the last ten years, Sir James Alderson, though still residing in London, must have been little known to most of those present here to-night; but, as a member of the Council of this Society when he was its President, I can speak of his gentlemanly appearance and bearing, of his polished manner, and of his cultivated style of address and conversation. No office lost honour or dignity in his keeping. He was a highly cultured physician, acute and reliable in diagnosis, careful and minute in treatment, and earnest and strict in the discharge of his duty, both to his patients and to his professional brethren.

Robert Wishart Lyell is the first and youngest of those deceased *resident* Fellows, to whose character, accomplishments, and labours I have now to advert, and whose loss to many of us is embittered by the rupture of the bonds of acquaintance, or the deeper ties of friendship. Born in London, he died, after a brief and unexpected illness, on the 2nd of last October, at the early age of thirty-three, having been a Fellow of the Society a short three years, but still having, in association with Dr. R. D. Powell, furnished a paper to our 'Transactions' "On Basic Cavity of the Lung treated by Paracentesis," vol. lxiii, 1880.

His early display of intellectual force at St. Olave's School, Southwark, where he was usually at the head of his classes, and subsequently at King's College, where, amongst other distinctions, he gained the Warneford Scholarship (1861), was fully maintained by his taking honours in nearly every subject at the M.B. Examination of the University of London in 1871, and by his graduating M.D. in the following year, also with honours. Thus equipped, he passed rapidly through the offices of

house Physician to the Seamen's Hospital at Greenwich, and of house Surgeon to the Royal Infirmary at Manchester, to reach the post of Surgical Registrar and tutor at the Middlesex Hospital (1874). Having already joined the Royal College of Surgeons of England as a Member in 1871, he became a Fellow of that College in 1875. Henceforth directing his energies to surgery, and especially to ophthalmic surgery, he was appointed, in quick succession, Ophthalmic Surgeon to the Great Northern Hospital, assistant Surgeon to the Middlesex Hospital, and also assistant Surgeon at the Royal London Ophthalmic Hospital in Moorfields.

Dr. Lyell did not live long enough to mature his observations and reflections sufficiently to enable him to publish much; but his reports as a surgical registrar are said to have been clear and full, and the assistance which he rendered to the Clinical Society as secretary to the Committee on Excision of the Hip, was very valuable: both instances of his ability afforded promise of good scientific and literary work. He was becoming known as a sound practitioner, and had already proved himself to be a painstaking and trustworthy teacher, always mastering the subject on which he was working, or which he was preparing for his lectures. The circumstances attending his sudden illness and death, intensified the regrets experienced by his colleagues, his pupils, and his friends. Whilst engaged in preparing the introductory lecture to be delivered at the commencement of the winter session at the Middlesex Hospital Medical School, and whilst studying the chemical affinities of bacteria, he was seized with acute pneumonia, and his speedy death on the very day on which the lecture was to have been given, threw a gloom over the opening ceremony, and deepened the sorrow felt by all at the premature close of so promising a career.

Alexander Silver, at the time of his death, on the 16th of July last, at the age of 41, had been eleven years a Fellow of the Society. Born in Forfarshire, his father

being, it would seem, a farmer of moderate pecuniary resources, he exhibited from his early youth the ambition, force, and perseverance, which so often characterise the sons of Scotland. Sufficiently successful at his parish school to obtain a bursary at Aberdeen, he coupled the pursuit of his medical studies at that University with the duties of a tutor; whilst in the summer months he worked on the family farm. Nevertheless, he took his degrees of M.A., M.D., and C.M., before he had reached the age of twenty-three. By assisting some of the Professors by giving private tuition, and by the compilation of a student's "Outlines of Elementary Botany" (which was his favourite science), he occupied the next few years at Aberdeen; when, at the age of twenty six, he was transferred to London, being appointed Lecturer on Botany at the London Hospital Medical School. In due time, Dr. Silver was chosen assistant Physician, Lecturer on Clinical Medicine, and then Physician to the Charing Cross Hospital, posts which he held up to the time of his un-anticipated death.

Besides the 'Outlines of Botany,' his literary exercises comprised the editing of the fourth edition of Mead's 'Manual of Practical Medicine' (1874), and the production of papers on the trial of G. Stephen, "On the use of Veratrum viride in Rheumatism," and, in conjunction with Mr. Barwell, on 'A Case of Left Lumbar Colotomy.' For the last sixteen years he had also been engaged on the staff of the 'Medical Times and Gazette,' having latterly been the Sub-editor of that Journal, in the pages of which a warm tribute of regard appeared from the pen of the Editor, with a kindly acknowledgment of the valuable services and personal qualities of Dr. Silver, as an able and industrious colleague and coadjutor.

Joseph Thomas Clover, for many years the foremost administrator of anæsthetics, and therefore the foremost minister of relief of human suffering and human dread of suffering, in metropolitan practice, died at the age of fifty-seven, from the effects of long-existing pulmonary disease.

He was a Fellow of the Society for twenty-nine years, and served on its Council in 1873.

A native of Aylsham, in Norfolk, he received his early education at Grey Friars School, in Norwich, and then became a pupil at the Norfolk and Norwich Hospitals, serving for two years as a dresser to the late Mr. Gibson. At University College, London, when yet a student in the medical classes, he was soon distinguished amongst his companions for his ability, diligence, and zeal, although, owing to his even then delicate health, he never competed for class honours. In the University College Hospital, he acquitted himself so satisfactorily in the various junior offices, from dresser and clerk to house surgeon and physician's assistant, that he was regarded as a most promising aspirant for the responsible position of Resident Medical Officer, to which he was elected when only twenty-three years of age. Mr. Syme, whose house surgeon Clover had been, would have gladly taken him to Edinburgh, but he declined that offer.

It was from this date that my personal knowledge of Clover began, and, I need hardly say, it continued until his death. In the hospital work he was indefatigable, often to the detriment of his frail constitution. Did affairs pursue their ordinary course, his management was such as to ensure the confidence of all concerned alike, namely, of the committee, the staff, and the pupils, of the nurses, patients, and domestics. Did any emergency arise, such as might be due to a cholera or other epidemic, or to a great railway accident, his powers of endurance rose to the occasion; for although he evidently felt the strain, his rule of conduct was *constancy to duty*. In his office, it fell to his share to administer anæsthetics, chloroform being at first the one employed; he also had to instruct others in their administration; and thus he acquired a large and full experience of the action of these potent agents, of the dangers associated with their use, and of the cautions to be observed in administering them. After five years of steady hospital work, and after having obtained

the Fellowship of the Royal College of Surgeons (1853), Clover entered upon surgical practice, fully resolved to pursue it generally, and already making good progress in it. But circumstances proved too strong for him, and his uncertain health combined to make him adopt the career of a special administrator of anæsthetics, in which capacity he so excelled, that his aid was sought on all hands, to suspend the consciousness of both bodily and mental pain of those who had to undergo the often prolonged operations of modern surgery.

In the *study* of anæsthetics, Clover was open-minded, and at once scientific and practical. He tested all and neglected none, being in this respect a worthy successor of Dr. Snow. He early perceived the importance of diluting chloroform vapour with air, in order to render it less dangerous; and he not only laid down a rule as to the proportion in which they should be mixed (a subject recently thought worthy of special experimental research by M. Paul Bert), but he invented first his bag and inflator, and afterwards his more portable and now generally used inhalers. He was selected as a delegate of this Society on the Committee appointed to study and report on the effects and mode of administering chloroform; and for his practical and valuable assistance he received the Society's thanks. The article on "Anæsthetics," published since his death in Dr. Quain's 'Dictionary of Medicine,' is of the highest order of merit. It discusses the properties and actions of all the known agents employed for producing anæsthesia, and it embodies the results of an altogether unrivalled experience, combined with the impartial conclusions of an unprejudiced mind.

In the *administration* of anæsthetics, Clover was rapid and certain, and so far safe that operators felt unusually relieved from anxiety whenever he was engaged. His manner was quiet, cheerful, and encouraging to the patient, and he was frequently very helpful to the surgeon. In the discharge of his duties as Anæsthetist to

the Dental Hospital and to the Westminster Hospital, and as Lecturer on Anæsthetics at University College Hospital, he was always delighted to explain his methods and views.

Clover's ingenuity was not limited to the invention of contrivances adapted to the administration of anæsthetics; but he had true surgical instincts, and devised several other instruments, especially a double-current catheter, and the elastic-ball and glass-bottle apparatus for washing the *débris* of crushed calculi from the bladder, an apparatus which has been adopted, though with various modifications, by others.

Clover was, as a man, modest, unselfish, and true in all his social relations. Though an invalid almost all his life, and latterly a victim to the wearisome and wearing effects of chronic pulmonary disease, he was always patient, resigned, and even cheerful. It is said of him by his friend and last medical adviser, Dr. Sidney Ringer, that "He was gentle, amiable, uncomplaining, and grateful to the last," and he adds, "The world wants one true man since he was taken away."

For myself, I can conscientiously state that, through a friendship lasting for more than thirty years, I never observed one single trait of character, nor one single action, which laid him open to censure; whilst, on the contrary, I can now recall numerous instances of his perfect sincerity, his invariable kindness, and of the pleasure which he felt in going about doing good in his vocation.

Robert Taylor, who died, in Portman Square, so recently as the 26th January in this year, entered the Society in 1852, and therefore enjoyed its Fellowship for more than thirty years. The son of a Dumfries-shire gentleman, he graduated at Edinburgh in 1841, became a member of the College of Surgeons of England in 1842, and a Fellow of that College in 1858.

He was formerly Surgeon to the Cripples' Home in Hill Street, Marylebone, and also to the Central London

Ophthalmic Hospital. Several papers by him, appeared in Journals or in Transactions. To the 'Medical Times,' he contributed papers on "Sympathetic Inflammation of the Eyeball," and "On Anæmic Protrusion of the Eyeball;" to the 'Pathological Transactions' communications "On the Corpora Amyloidea in the Lens," and on "Cataracts;" and to 'Beale's Archives' a joint paper with Mr.E. C. Hulme, "On Cases illustrating the use of the Ophthalmoscope."

Dr. Taylor was of singularly retired habits, and apparently much isolated from his professional brethren; but up to the last few weeks of his life he was an habitual frequenter of the Society's reading room, thus keeping alive his evident interest in the affairs of the profession itself.

George Critchett, the well-known ophthalmic Surgeon, whose death after a short illness, on the 1st November last, took all but his most intimate friends by surprise, was a Fellow of this Society for thirty-five years, having joined it in 1847, served on its Council in 1865, acted as a Referee between 1867 and 1871, and been a Vice-President in 1872. He contributed to the 'Transactions,' an interesting paper on an "Operation for Congenital Cataract in an Adult, followed by Division of the Recti Muscles, for the purpose of controlling the Oscillation of the Globes" (1855).

A native of Highgate (1817), he commenced and completed his professional studies at the London Hospital Medical School, having been a pupil of Mr. Scott. He became a member of the Royal College of Surgeons in 1839, and a Fellow by examination in 1844. He was very early chosen as Demonstrator of anatomy in his School and assistant Surgeon to the Hospital, in which he was subsequently promoted to be full Surgeon. During the fifteen years in which he held these hospital appointments, he proved his capacity as a sound surgeon and a most dexterous operator. But he already began to show a leaning towards ophthalmic surgery, by giving lectures on that subject at his own school; after a time he was appointed

in succession assistant Surgeon, Surgeon, and Consulting Surgeon to the Royal Ophthalmic Hospital in Moorfields, and also Ophthalmic Surgeon to the Middlesex Hospital. By a series of publications of lectures and other essays, he showed his strong attachment to his chosen speciality; and he suggested several new operative proceedings. He published a course of lectures on "Diseases of the Eye" in the columns of the 'Lancet;' also a paper entitled "Practical Remarks on Strabismus;" the description of a "Mode of forming Artificial Pupil by Iridesis, or Ligature of the Iris" (a method, however, which he afterwards abandoned); an account of the "Operation for Strabismus by the Subconjunctival method, with hook and scissors;" a paper on "Linear Extraction of Cataract," read at Heidelberg; another on "A mode of Enucleating the Eyeball;" and another on "Superficial Affections of the Eye," read at a meeting of the British Medical Association. His views on the pathology and treatment of eye diseases and injury were based on a large experience, of which he was always able to avail himself.

As a lecturer and clinical teacher, his voice and manner, as well as his matter, at once arrested attention; whilst by his acknowledged dexterity—which could hardly be surpassed—he may be said to have initiated a style of manipulation which has left its mark on the practice of his pupils and successors.

The eminence which he had attained in the profession, and the regard in which he was held, were evidenced by the various honorary offices and titles which were conferred upon him. He was for seven years a diligent member of the Council of the Royal College of Surgeons; he was also President of the Hunterian Society, President of the International Congress of Ophthalmologists, held in London in 1872, Vice-President of the Ophthalmological Section of the International Medical Congress of 1881; and, up to the time of his death, Vice-President of the recently formed Ophthalmological Society of the United Kingdom. He was also a member of the Medical Society of Louvain,

an honorary member of the Academy of Medicine in Brussels, and member of the Imperial Academy at Rio.

In consultation he was prompt, yet taking infinite pains, —decided, but never off his guard. To his patients he was most considerate, and he invariably inspired them with confidence. To his friends, and he had many, he was cordial and hospitable. His unexpected death came to most of them as a shock, and they will long remember his kindly grasp and greeting.

Thomas Bevill Peacock, to whom a special fate brought death in a ward of the hospital with which he had long been connected, was for no less than thirty-seven years a Fellow of this Society, in the service of which he had acted as a Member of the Council, on the Library Committee (1855), as Secretary (1855-6), as Referee (1857-65), and as Vice-President (1867). To the 'Transactions' he very early contributed two excellent papers, on "A Case of Malformation of the Heart" (1847), thus early indicating a choice of subject for research, to which he ever after remained faithful.

Born at York in 1812, of Quaker parentage, his father being a merchant in that city, he was destined to submit to many changes of residence, and to be occupied in many fields of labour. Thus, he was apprenticed to a member of the Fothergill family in practice at Darlington, and his London education took place partly at University College and partly at St. George's Hospital. He first joined the ranks of our profession as a member of the College of Surgeons of England (1835). He then went two voyages to Ceylon (1835-36); next he became house Surgeon to the Chester Infirmary, a post which he held for four years; and then he appeared in Edinburgh, where, turning his attention henceforth to Medicine, he acted as house Physician to the Infirmary, and devoted himself specially to the study of morbid anatomy. He took his degree of M.D. Ed. in 1842. Somewhere about this period he also visited the Paris Schools, in which doubtless his predisposition for pathological investigations was still further confirmed. In

1844, he attached himself to the College of Physicians of London as a Member, and in 1850 he became a Fellow. It was in the interval between these two dates, that he first began to display the amount and character of the materials he had collected, and the special exactitude of his work, for he published in quick succession, besides his two preliminary essays in our 'Transactions' already mentioned, the four following papers in them :—"On the Coexistence of Granular Disease of the Kidneys with Pulmonary Consumption" (1845); "Tables of Weights of some of the Organs of the Body" (1846), a much consulted and often-quoted paper; "On the Influenza or Epidemic Catarrhal Fever of 1847-8" (1848); and "On Aneurism of the Coronary Artery" (1849).

Though now and henceforth settled in London, his destiny to wander still pursued him; for we find him proceeding from one institution to another, each change, however, advancing him in professional status, and giving him greater prominence and reputation. Thus, he was at the same or at different times Physician to the Royal Free Hospital, and then Physician and afterwards Consulting Physician to the Victoria Park Hospital for Diseases of the Chest, an institution which sprang out of a Dispensary, and both of which Charities were supported by himself and by his friends. But his chief appointments were at St. Thomas's Hospital and Medical School, with which he was actively connected, as assistant Physician and Physician for twenty-eight years, from 1849 to 1877, since which latter date and up to the time of his death, he ranked as Consulting Physician. In the School, he was at first Lecturer on Materia Medica, and afterwards Lecturer on Medicine. During this long period, his additions to medical literature, and it should be added to medical knowledge, were again constant and substantial.

Thus, he published an elaborate paper on "The Weight and Dimensions of the Heart in Health and Disease" (1854), "Lecture on the Varieties of Continued Fever," (1856), his chief and most important work on "Malformation of the

Human Heart," of which two editions were published (1858 and 1866), a paper "On the Diseases of Metalliferous Workers," his Croonian lecture "On some of the Causes and Effects of Valvular Disease of the Heart" (1865), and comparatively recently an essay "On Prognosis in Valvular Disease of the Heart" (1877). Besides these, he contributed several papers on the "Pathology of the Heart" to the Edinburgh Monthly Journal, articles on the "Weight of the Heart," and on "Aneurism of the Heart," to 'Reynolds' System of Medicine,' Statistic or Anthropological papers to the 'St. Thomas's Hospital Reports,' and lastly, upwards of 150 specimens with descriptions, to the meetings and 'Transactions of the Pathological Society,' of which, indeed, he was one of the most zealous members, serving in turn as Secretary, member of Council, and President.

The amount of time and labour which Dr. Peacock expended in accumulating and arranging his facts, must have been prodigious, involving the collecting, weighing, measuring, dissecting, and preserving so many separate specimens. It is probable that he made with his own hands as many pathological preparations as any one of his contemporaries, even any one museum curator. Possessed of the true Hunterian spirit, and no doubt solicitous for the permanent safety of his collection, it was not a matter of surprise that, turning to the College of Surgeons, whence he had obtained his first diploma, and in which he had officiated as the first Examiner in Medicine (1868), he should have presented his numerous and unrivalled examples of Cardiac diseases and malformations to the Hunterian Museum in that institution, a donation for which he received not only the thanks of the Council, but the Honorary Gold Medal, which is rarely conferred by the College, as its highest mark of distinction, in recognition of special services rendered to it or to science.

It was but a few hours after he had listened at that College in February, 1877, to Sir James Paget's Hunterian

Oration—at which I sat next to and afterwards conversed with him—that Dr. Peacock had his first warning of paralysis, which occasioned his enforced retirement from hospital work, and, indeed, from active private practice likewise; and although he continued to appear at the meetings of a few societies, and superintended some publishing work he was obviously enfeebled, and lost his characteristic power of sustained effort. On the last day of May, 1882, he was escorting some friends through the pavilions of St. Thomas's Hospital, when he was again seized with paralysis, and this time fatally; for having become unconscious he was carried into one of his own former private wards, and expired at an early hour of the first June morning, in the seventieth year of his continuously laborious life.

I venture to use this latter phrase, because I think that whoever contemplated his strongly marked individuality both of person and of character, could scarcely help feeling that, from his earliest youth, he must have been in all respects very similar to what he was in after years. Slight in figure, serious or even grave in expression, quiet and undemonstrative in manner, he was, one would imagine, as painstaking and laborious as a boy, a scholar, and an apprentice, as he subsequently became as a student, an investigator, a teacher, and a Physician. The extent of his anatomical collections showed his love of facts, whilst his addiction to numerical estimates proved his love of accuracy in the appreciation of them. Even in his pleasures he was precise and methodical; for in his recreative incursions into the field of Ethnology, his observations and inferences were accepted with confidence; and in his frequent journeys abroad he is said to have devoted himself with success to the acquisition of a sound judgment and cultivated taste in the domain of art.

He has left behind him the reputation of a simple, conscientious, and kindly man; of an eminent pathological anatomist; of a serious and accurate teacher; of a learned Physician; and of a sound practitioner.

Sir Thomas Watson, Bart., the last and, as all will freely admit, the most distinguished of our deceased resident Fellows, the veteran Physician, the Nestor of English Medicine, died at his son's residence near Reigate, on the 11th of last December, at the patriarchal age of ninety years and nine months.

The descendant of a Northumbrian family, he was born at Montrath, near Cullompton, in Devonshire; his birth is duly registered at the neighbouring parish of Kentisbeare, as having taken place on the 7th March, 1792. He received his grammar-school education at Bury St. Edmund's, and afterwards proceeded as a pensioner to St. John's College, Cambridge, where he came out tenth in the Wrangler's list for 1815. He was made a Fellow of his College in 1816, and took his degree of Master of Arts in 1818. Owing to a rule which enabled one Fellow to study Medicine away from the College, he retained his Fellowship, and during the eight or nine years of its continuance he was partly working at St. Bartholomew's Hospital under Abernethy (1819), and afterwards attended the medical classes for one session in Edinburgh. He then returned to Cambridge, and even took his share of duty as a junior proctor; at length he graduated as M.D. in 1825, when he was already thirty-three, at which time he married and gave up his Fellowship.

Proceeding next to London he became, in 1826, a Fellow of the London College of Physicians, and in the following year was chosen Physician to the Middlesex Hospital on the resignation of Dr. Southey. This post he held until the year 1843; and it was during those sixteen years that he developed the qualities which immediately and for long afterwards brought him increasing distinction. Thus, from 1828 to 1831, he was Professor of Clinical Medicine in the newly-founded University of London, now University College, delivering his lectures, however, at the Middlesex Hospital. In 1831 he resigned that office to take the Professorship of Forensic Medicine

at King's College, from which Chair he was moved to that of Medicine in the same institution, as the successor to Dr. Francis Hawkins. After holding this appointment for four years, he resigned it in 1840, in preference to severing his connection with the Middlesex Hospital as one of its Physicians. At a later period he was nominated consulting Physician to King's College Hospital. Finally, in 1843, owing to his increasing practice, which at first gathering slowly, had then become very large and onerous, he also resigned his post as Physician at the Middlesex Hospital.

It was after Dr. Watson's retirement from the Medical Professorship at King's College, that his course of lectures on "The Principles and Practice of Medicine" appeared in the 'Medical Times and Gazette,' a Journal to which he had contributed several previous essays and discourses. These lectures occupied two years in publication (1840-2), appearing weekly in four volumes of the journal, xxviii to xxx. Their reception was so favorable that they were published separately in 1843; since that date four other editions have been called for, viz. in 1845, 1848, 1857, and 1871, the later ones having been carefully revised, and the last one being especially noticeable for containing evidence of their author's candour in his abandonment of views which he thought no longer tenable. The estimation in which these celebrated lectures, Watson's "Magnum opus" have always been held, is quite remarkable, and, in our time, unique. They possess all the advantages of having emanated from one wise, observant, and evenly balanced mind; whilst the extensive and profound knowledge of disease, as it had been actually observed and carefully noted by the author himself, not only secured them from error, but imparted to them a rare combination of originality, accuracy, and force. His descriptions are so full and clear that, in this respect, Watson has been compared with Sydenham; and his style is so scholarly, that it has gained for him the appellation of the Cicero of English Medical Literature; whilst his apt illustrations, his special

touches, now sad, now humourous, lend a peculiar quality to his writings, many a chapter of which, when once commenced, is sure to hold the attention of the reader to its end.

In connection with the Royal College of Physicians, which, as just mentioned, he had joined as a Fellow in 1826, Dr. Watson was Gulstonian Lecturer in 1827; he delivered the Lumleian Lectures in 1830-31; and was Lecturer on Materia Medica from 1833 to 1835. He was Censor in 1828, 1837, and 1838; on the Council of the College from 1836 to 1868; its representative on the General Medical Council from 1858 to 1860; and, finally, at the age of seventy, and after forty-one years of association with it, he became its President. This latter high office he held for five years, from 1862 to 1867. In this distinguished position his conduct of affairs was perfect, being ever characterised by a quiet and sedulous attention to business, great common sense, carefully considered and powerful addresses, invariable courtesy to others, and constant solicitude for the welfare of the College, in the Censor's room of which his admirable portrait by George Richmond, preserves the remembrance of his intelligent, kind, and manly face.

The unquestioned confidence placed in Sir Thomas Watson by the profession and the public, was testified by his very large practice; by his appointment in 1859 as Physician Extraordinary to Her Majesty; by his being called, in 1861, to share the grave responsibilities involved in the fatal illness of the late Prince Consort; by his receiving, during the latter part of his Presidentship of the College of Physicians, a Baronetcy from the Crown (1866); and by his being made Physician in Ordinary to the Queen (1870).

Numerous other honours and other duties also fell to his lot. He became a Fellow of the Royal Society in 1859; Honorary Fellow of his old college at St. John's, Cambridge, to which he was elected together with Sir John Herschel in 1862; Doctor of Laws, Cambridge, in

1864; and Honorary Fellow of the King's and Queen's College of Physicians, Ireland.

In our own Society he was a Fellow of forty-five years' standing, and during his connection with it had been on the Council for three years (1840-1 and 1852), had acted as Referee during four years (1842-3, 1847-9), was a Vice-President for two years (1845-6), and would undoubtedly have been President, had his numerous avocations permitted him to undertake the office. As a proof of his continued interest in any new movement, he consented to act as President of the Pathological Society (1868), and subsequently of the Clinical Society also.

But all his titles must yield in significance to that of being termed "the greatest English Physician of the present century," a designation which he fairly earned by his professional eminence, his moral worth, his sagacity, his integrity, his undeviating regard for the feelings and interests of his brethren, and by his steady adhesion to those high principles of duty, which he so eloquently expounded in his well-known and often-quoted "Introductory Lecture," and which made him so fit an adviser on ethical questions relating to our profession.

To some men it happens that extreme old age brings opportunities for the display of petulance, or for the commission of error, or, it may be, for the opposite alternatives of senile inability, inaction, and lapse into oblivion; but it was not thus with Sir Thomas Watson. At three score years and ten he was unanimously welcomed as President of his college, at four score years he was allowed, unwillingly, to withdraw from the absorbing work of our profession, and even then, during the next decade of his long and honoured career, he emerged occasionally from his retirement to show his interest on some special occasions, at some Society, Association or Congress, or at an assembly of the Fellows of his own College. It is recorded that even as late as last March, when he had completed his ninetieth year, he took a leading part in the ceremony of re-electing the President of that College, amidst mani-

festations of respect from the assembled Fellows, all of whom rose as he advanced in the absence of the senior Censor to hand over the insignia of the presidential office to Sir William Jenner. The crises and trials of his last illness were watched with anxiety and sorrow, and his death was followed by notices which seemed to exhaust the language of eulogy; for whilst time had not dimmed his fame, it had served to change respect into affection, and admiration into reverence.

An old friend of Sir Thomas Watson, and a former warm-hearted president of this Society, in a communication from Nice, says, "I have thought how best in one short sentence to write his epitaph. I can find no fitter words than those written well nigh 3000 years ago. 'Mark the perfect man, and behold the upright, for the end of that man is peace.'" *

It now only remains for me to mention two deceased Honorary Fellows, the one a physicist, chemist, and physiologist, the other a great naturalist. They were labourers in fields situated on the borders of the domain of Medicine and Surgery proper. They were eminent representatives of a class, which both gives and receives honour by being enrolled amongst us; and although the death of such men may not affect us so profoundly as that of our more intimately known professional brethren, yet it leaves a sense of vacancy and loss which it is our duty to acknowledge, with some accompanying homage to their worth.

James William Draper, of New York, though justly

* I once, whilst yet a student, consulted Sir Thomas Watson, then Dr. Watson, as to a persistent, dull pain through the upper part of the right chest, from front to back. Looking very grave the while, he took infinite pains in examining me, and entered the details in one of his little, fat, red-covered case books. When he had finished, he laid his hand on my right shoulder and said kindly, "If you don't trouble yourself about that, it will never trouble you; if it should, come back to me." "But," he added, "you will outlive me." I afterwards found that the pain was due to, and kept up by, the use of the lesser pectoral muscle in the frequent mounting of omnibuses, to which I was somewhat addicted in those days.

held to be an American philosopher, for he was early naturalised in the United States, was an Englishman by birth, and remained in this his native country until he was twenty-two years of age. He was born at St. Helen's, in Lancashire, in 1811, his father, the Rev. J. C. Draper, being a Nonconformist Minister. His early training took place at a neighbouring Wesleyan school at Woodham Green, in which institution he is said to have acquired his first taste for science. His higher education was continued and indeed nearly completed at the London University, now University College, where I find, on searching the records for 1829-30, when his father had apparently moved to Sheerness, his name is entered as a student in the classes of Jurisprudence and English law; and, where, in 1830-31, he is indicated as having joined the class of Chemistry, then under the charge of Professor Turner—this time his residence being given at No. 9, Old Jewry. It is more than probable that it was to the teaching and example of his distinguished master, that Dr. Draper owed his subsequent preference for chemical physics, and the chemical and physical aspects of physiology; for it is well known that Turner, in his lectures and writings, paid unusual attention to those subjects.

Just half a century ago and, as already said, at the age of twenty-two, young Draper followed his family to America, which henceforth became his adopted country. In 1836, he graduated M.D. at the University of Pennsylvania, his thesis on that occasion "On the Crystalisation of Camphor under the Influence of Light and Radiant Energy," being selected for publication. Doubtless owing to the merit exhibited in this, his first essay in scientific research, he was, in the same year, when only twenty-five years of age, appointed Professor of Chemistry and Physiology in Hampden-Sidney College, Virginia; and from thence, in consequence of his further display of scientific ability, he was promoted three years later to the Chair of Anatomy and Physiology in the University of New York which appointment he held through the remainder of his

active career. He assisted in establishing the University Medical College in that City, and for a long time presided over the Medical faculty of the University, from which sphere of activity he retired in 1876. He was elected an Honorary Fellow of this Society about seven years since.

Dr. Draper's original investigations, extending over a period of forty years, embraced many problems in chemical physics and physiological chemistry. The papers on these subjects appeared originally in American Journals or in the 'London Philosophical Magazine,' and were collated and published by him in 1878, in a volume of scientific Memoirs, containing thirty separate essays. His larger work on 'Human Physiology,' published originally in 1836, and of which a second edition appeared in 1858, exhibits his tendency to regard the phenomena of life from a statical and dynamical point of view, as, indeed, was natural in one devoted to his special lines of inquiry. It clearly establishes his claim to an advanced position amongst the original physiologists of his day.

From a physicist he became a philosopher, and directing his attention to the great problems concerning human progress, he put forth two well-known works, in one of which he traced "The History of the Intellectual Development of Europe," and in the other "The History of the Conflict of Religion and Science." In alluding to the transference of his own thoughts from physical to philosophical questions, he says: "That he recognised how they have been interwoven, each preparing the way for its successor." "Is it not true," he characteristically asks, "that for every person the course of life is along the line of least resistance, and that in this the movement of humanity is like the movement of material bodies?" a question which, it may be remarked, will be regarded as involving a mere truism, a valuable axiom, or a dangerous dogma, by different persons, according to the views they may hold in reference to the notion of free will. In Dr. Draper's bold and unflinching exposure of past and present antagonisms between religion and science, in which he

shows no indifference to the former, may be traced perhaps the outcome of his early Nonconformist training, mingled with the effects of his subsequent ardent devotion to severe scientific work.

After his retirement from his position at the University of New York, Dr. Draper resided on the banks of the Hudson, a few miles from that city. Here he enjoyed a well-earned repose in the Society of his distinguished sons, gratified by the fact of his more important works having been translated into many European and Asiatic languages. He died on January 4th, 1882, and was buried in Long Island amidst marked demonstrations of respect and regard from numerous friends, colleagues, and delegates from distant scientific societies.

Charles Darwin, THE Naturalist, as he may with emphasis be entitled, occupied so lofty and conspicuous a position in the Pantheon of science, and the Theory to which his name will ever be attached, has been so engrafted into the stock of knowledge and the intellectual training of the day, that it is unnecessary, and would be unseemly, if I were to enter into many minute details concerning the well-known life and labours of our illustrious countryman. But, as one of our most distinguished Honorary Fellows, silence concerning him would even be more culpable.

Born at Shrewsbury on the 12th February, 1809, the son of a Physician in that town, we may suppose him to have inherited, from both parental lines, powers of observation, reflection, and imagination of no ordinary strength, together with a love of nature so deep as to command his life-long attachment to the contemplation of her ways; for his mother was the daughter of Josiah Wedgewood, and his grandfather was Erasmus Darwin, the author of the 'Botanic Garden,' 'Phytologia,' and 'Zoonomia.' From the Grammar School at Shrewsbury, he repaired to the University of Edinburgh to study Medicine, and here, as a mere tyro, he showed his love of natural history and biology by some observations "On the Ova of Flustra." After

two years' training in the fundamental sciences required for a medical degree, years which we may be sure were well spent, he left Edinburgh and the pursuit of Medicine. Proceeding then to Cambridge he graduated in 1831, and immediately after, when only twenty-two years of age, through the intervention of his friend and teacher, Professor Henslow, he accepted the office of Naturalist in the celebrated expedition of the "Beagle," under Captain Fitzroy. This occupied him five consecutive years (from 1831 to 1836), during which period he suffered much from sea-sickness and from other inconveniences and trials to his health. The extraordinarily prolific results of his botanical, zoological, and geological observations made during this voyage, only gradually became known. An immense collection of specimens of plants, of living and extinct animals, and also of minerals, furnished new materials for Monographs or papers by Owen, Bell, Gould, Jenyns, and Waterhouse, as well as for an exhaustive Monograph by himself on the 'Cirrhipeds and Allied Animals;' whilst an equally remarkable accumulation of recorded observations of natural phenomena of the most varied kind, gave a special value and interest to his own 'Account of the Natural History and Geology of the Voyage of the Beagle,' and to his charming exposition of the 'Structure and Distribution of Coral Reefs and Volcanic Islands.'

It appears certain that already Charles Darwin's thoughts were brooding over the great problems of the origin and succession of life upon the earth, problems the attempt to solve which occupied the next twenty years of his life. Familiar with the poetic imaginings of his grandfather, the striking fancies of Lucretius, and the speculations of Lamarck, Buffon, and others, it is certain that he very early caught glimpses of the possible modes of action of natural causes in the production and perpetuation of variations of form and structure in plants and animals during the lapse of ages. But for twenty years he waited, accumulating facts, from authorities old and recent, from the

experiences of the breeders of animals, and from the results of his own ingeniously devised experiments,—all which facts he co-ordinated in his own masterly manner, and finally deduced from them the several principles which underlie his great Theory. It was only when it was announced that Mr. Wallace had arrived at similar general conclusions, that Darwin consented to expound his views publicly, and the papers of both Naturalists were read at the Linnæan Society on the same evening, 1st July, 1858. There is, it seems to me, a grandeur in this enforced silence, this prolonged self-restraint. But all the more decisive was the stroke which, thus deliberately delivered by the fact-laden hammer and logical force of this scientific Thor, finally shattered the old notion of the independent creation of species (hundreds of thousands in number), a notion, however, by that time rather tolerated than accepted, difficult to conceive as true, and leaving unexplained the multitude of likenesses and differences of development and structure, with which the organic world both past and present abounds.

That greatest biological work of our age, 'The Origin of Species,' appeared in 1859; it ran through many editions, it appeared in many languages, and it was hotly criticised; but its triumphant "coming of age" was celebrated in 1880, by an admiring and ardent supporter, Professor Huxley, in an eloquent and well-remembered discourse delivered at the Royal Institution.

It is needless here to mention Darwin's subsequent publications. They are chiefly devoted to further explanations of his views, and to a fuller exposition of his rich accumulation of experiments and observations. All of them exhibit his marvellous knowledge of, and insight into, the operations of nature, and disclose incidents of plant and animal life so wonderful as to be read of with delight. One work only, 'The Descent of Man,' raised a storm of opposition, both metaphysical and theological. But, in the meantime, the evolutionary hypothesis, previously vague and unsubstantiated, had become conditioned

and workable as a Theory, which proved to be fruitful beyond measure. Resting on those brilliant and incontrovertible generalisations, the inherent "tendency of organisms to vary," the perpetual "struggle for existence," the "survival of the fittest," "modification by descent," "natural selection," "sexual selection," co-relation of parts and organs, "inheritance," "atavism," and "reversion" —each supported by hosts of facts—the Darwinian philosophy came to be accepted by some, tacitly admitted by many, adopted, with reservations, by others, and rejected only by a few. The doctrine of evolution has gained many disciples amongst the foremost men of the age; and these have, by its suggestiveness, made many discoveries, and solved various intricate questions as to structural affinities, modifications, adaptations, and mimicries of form and colour, noticeable in plants and animals. But its application has not been limited to botany, zoology, and comparative anatomy, for its influence has permeated almost every branch of science and nearly every sort of inquiry. It has acted, as it were, like a ferment, stimulating simultaneous or subsequent investigation in new directions, breaking up old, and creating new combinations of thought, in history, sociology, moral and intellectual philosophy, and philology, in short, in almost every department of human knowledge and research.

Perhaps no one man has ever exercised greater influence over his contemporaries than Charles Darwin, and no one has ever been more freely granted a sort of scientific sovereignty. He was made a Fellow or Member of every leading Academy or Society in Europe and America. The dread which his views once inspired has subsided. It has been recognised that what is true in them must stand; and the bitterness of the controversy concerning them was felt to be assuaged when, after his death, which occurred on the 19th April last, at his home at Down, in Kent, his remains were deposited in Westminster Abbey, amidst the ceremonials of religion and the reverence of a vast crowd of representative men of all classes and callings.

As members of the Medical profession we may indulge in the flattery of a belief that Charles Darwin, the neophyte Medical student at Edinburgh, acquired there such a knowledge of anatomy and physiology as helped materially in the work of his life; we must also acknowledge that not only in anatomy and physiology, but in pathology also, the great principle of evolution has already found a place. As members of this Society, we may be glad to remember that it was so far back as 1868, when disputations concerning his opinions were still rife, that he was elected an Honorary Fellow; and we may be proud to reflect that so illustrious a man has belonged to us for fourteen years, during the height of his world-wide fame.

I have now, gentlemen, completed these brief and imperfect valedictory notices, which, of course, do not lay claim to the exhaustive and critical characters of biographies. In regretfully bidding adieu to those who are no more numbered amongst us, what consolation, what lesson, may we derive from the history of their careers?

The thought has often occurred to me, and, no doubt, to many besides—"what an amount of accumulated experience and wisdom is blotted out at the death of each Master of the crafts of Medicine and Surgery—and if we reflect on the number of gifted, learned, and industrious men who have passed from our ranks during the last twelve months, we may well feel dismay that so much slowly acquired individual knowledge has been here extinguished, and so much personal power has thus died out. But fortunately for mankind, owing to the liberal intercommunication of ideas which distinguishes the true from the false disciples of Æsculapius, amongst the instruments of which intercourse are Societies or brotherhoods like ours, such knowledge and power are handed on from period to period, and the examples of one generation are emulated by its successors.

If we study the record of the lives of our deceased Fellows which I have put before you, we find that they possess one common characteristic, viz. *devotion to work.* Whether they were born to affluence, or entered upon life supported by moderate or scanty resources; whether their early education was of the highest order, or of humbler pretensions—provided for them, or secured with difficulty; at whatever medical school they were trained, whatever professional titles they acquired, whatever the branch of practice to which they had recourse, and, whether in London, in the provinces, or abroad; whether they had the early support of powerful friends, or struggled upward by themselves; whether they were shaped by circumstances, or compelled circumstances to bend to them; whether they aimed solely at professional eminence, or sought relief to their redundant energy in the duties of citizens besides; whether they were rewarded by the rich or the poor, by the public, the profession, the Government, or the Crown; and lastly, whether their lives were so prolonged as to secure the full fruition of their aims, or were cut so short that they saw but visions of future success;—it is apparent, I say, that all were devoted to their work.

Of the *character* of that work, burdened as it must have been with all the responsibilities of our anxious profession, perhaps with the troubles and vexations, the trials and failings of private life, from which no human being can escape, and associated with the strictest demand for integrity of character and regard for the interests of patients and of professional brethren,—I need not speak to you; and as to its *amount*, I may appeal to the sum of effort, which it has fallen to me to describe to night.

With regard to this work in reference to the special interests of this Society, which, however, are of minor importance in comparison with those of the public and of the profession at large, it may yet be well to add that, setting aside our deceased non-resident Fellows, who were ineligible for office in the Society, there remain in our list of deaths eleven resident or once resident Fellows.

Of these latter, six have served on the Council, four have acted as Referees, one has done good service on a Special Committee, two have been members of the Library Committee, two have been Secretaries, one has been a Treasurer, five have been Vice-Presidents, and one has been President, whilst five of the resident Fellows have contributed twelve papers to our 'Transactions.'

It has frequently been remarked, both in prose and verse, that the occasional contemplation of the lives and work of great and good men may help to encourage us to corresponding efforts; and perhaps I may venture to hope that the picture which I have endeavoured to present to you in this passing hour, of the weighty deeds of our recently deceased associates, may serve to deepen our convictions of the value and needs of our noble profession, may nerve such of us as are able to fresh exertions, and concurrently aid in advancing the interests and increasing the prosperity of this Society.

ON DISLOCATION OF THE FOOT

WITH

VERSION AND TORSION OF THE ASTRAGALUS.

BY

RICHARD BARWELL, F.R.C.S.,

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(Received May 23rd—Read October 24th, 1882.)

MANY instances of entire dislocation of the astragalus from its connections with the rest of the tarsus, and also from the bones of the leg have been recorded; it is to be noticed that in these cases the bone is very rarely projected straight out of the socket, but receives a twist so that its axes lie, and its surfaces look, in quite abnormal directions. There is, however, another form of injury in which the astragalus is likewise twisted or turned over, but remains in the tibio-peroneal socket, the bone is *in sitû*, but its axes and surfaces are misplaced, while its connection with the rest of the foot is sundered. This condition is extremely rare; moreover most of the few instances on record are described in so cursory and indefinite a manner that it is often doubtful what kind of injury is in reality indicated, and it is generally quite impossible to gain any idea as to its distinctive symptoms. The case, which I have now the honour to report to the

Society, presented itself under circumstances which enabled me very clearly to note its clinical features; while the operative treatment employed afforded an opportunity, as perfect as a post-mortem dissection, of verifying my previous diagnosis. I would hope, therefore, that some minuteness of detail in description will not be considered tedious.

George F—, aged 28, a coachman, of apparently healthy constitution, was admitted under my care into Charing Cross Hospital on 15th November, 1881, having received an injury to the right ankle. The accident occurred through the breaking of an axle, the gig fell over on its right side, and he was thrown out. The horse ran away, and the man holding the bridle, was dragged for some distance along the ground. He does not think this latter circumstance caused the injury, but believes that the foot was hurt when the gig fell.

I saw him about an hour and a half after the accident. The foot was greatly inverted, the sole looking almost directly inward, its front somewhat turned in, the heel raised. The inner malleolus was much hidden; beneath it the skin was thrown into two ridges by three deep folds drawn in segments of concentric circles from a centre a little above the malleolus. The cutis of this part was so much compressed that the ridges felt hard, tight, and unyielding. The outer malleolus projected abnormally, the skin over it rather tightly drawn. About an inch in front of it and a little below its level was a rounded projection, which also somewhat stretched the skin: An inch and a half up the leg and in front of the fibula, was a small but deep wound, from which venous blood flowed pretty freely. The man was unable to move the foot in any direction, and suffered a good deal of pain.

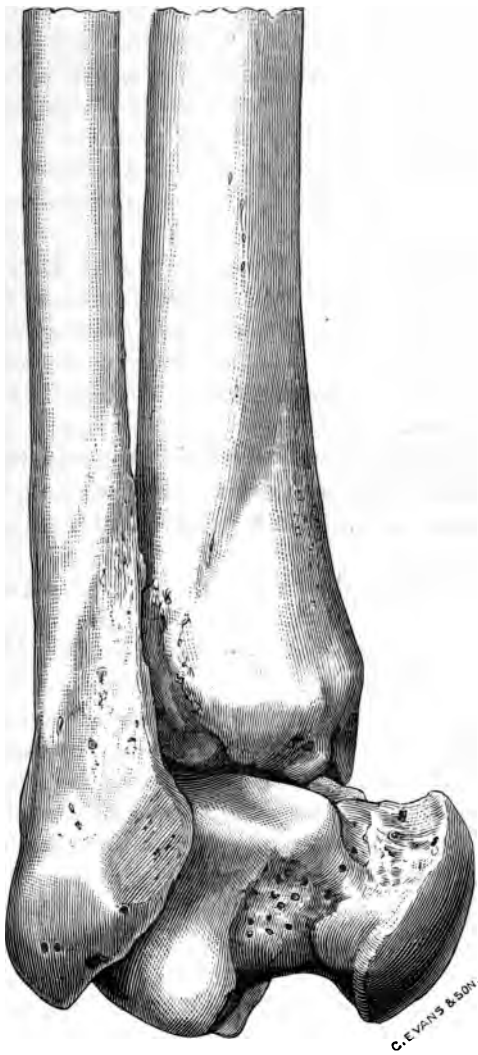
On palpation the following additional particulars could be made out. Below and in front of the inner malleolus deep pressure revealed absence of the usual bony substratum, the finger sank into a hollow, bounded in front by the tuberosity of the scaphoid, which lay abnormally

near the malleolus. The rounded projection in front of the outer malleolus could readily be recognised as the head of the astragalus. A little way behind this was a ridge of bone, also evidently a part of the astragalus, it led from the head backward and a little upward, disappearing under the upper part of the malleolus, at the angle between it and the horizontal anterior edge of the tibia. This ridge was markedly convex *outward*. The extensor tendons, pressed together, ran in a bundle a little distance inside the rounded projection. The wound communicated with the injury. No fracture of bone could be detected. The foot, incapable of active movement was hardly amenable, unless much force had been used, to any passive motion.

From these data the diagnosis was not difficult up to a certain point; namely, it was clear that the other bones of the tarsus were displaced from the astragalus. At this point, however, the niceties of diagnosis really began, for the astragalus, though evidently not in normal position, was certainly not out of its proper place. I therefore expressed to my class this view deduced in the manner described at p. 47, that the astragalus was twisted in but was not dislodged from the tibio-peroneal socket. Nevertheless, it seemed right to attempt reduction; therefore chloroform was given and, assisted by Mr. Morgan, I endeavoured in all ways to replace the bones, even Cline's method, of placing the knee against the prominent malleolus and forcibly drawing the foot in the direction contrary to its abnormal posture, was used. No effect was produced save some temporary change in the position of the foot. I then, rather in deference to the opinions of my colleagues than because I expected any better result, divided the tendo Achillis and renewed my efforts at reduction, but with like negative result. I ordered a paroplastic splint to be moulded on the limb in its then position; the wound to be injected with a solution of carbolic acid, and the same material to be kept applied on a suitable pledget.

November 17th.—The man having consented to the course I proposed, ether was administered, and I made a semilunar incision from the middle of the lower end of the tibia to the tip of the outer malleolus; the middle of this cut ran over the displaced head of the astragalus. The flap was turned up and the bone fully exposed. It was a little turned on its perpendicular axis, for its long axis ran from behind forward and a little outward; its head lying some distance in front of the outer malleolus. It was twisted ninety degrees or a quarter of the circle on its antero-posterior axis, its trochlea looking outwards, was in contact with the articulating cartilage of the outer malleolus, its inner surface rested upon the horizontal joint-surface of the tibia; its lower surface, that for the calcaneum, was opposite the articular facet of the internal malleolus; its outer surface faced the calcaneum. I noted especially how closely the inner upper angle of the trochlea fitted into the entering angle formed by the lower articulating surface of the tibia and the outer malleolus. The bone was not at all displaced forwards; that is, it did not protrude abnormally from the socket. The extensor tendons and the anterior tibial artery were all to the inner side. I did not see the peroneus tertius; it can hardly have been pressed inwards, but it certainly was not divided with the knife.

Having verified these particulars, I seized the bone in lion forceps, and, turning it still further in the same direction, divided what ligamentous fibres restrained that movement; these were not many, some of the posterior and a few of the internal lateral ligaments only required the use of the knife. The calcaneo-astragaloid interosseous ligament had been torn through. The bone, therefore, came away with singular ease and the bleeding was trifling. I then passed my finger into the large cavity; there was not the slightest roughness; no chipping of bony edges nor, as far as I could make out, any detachment of cartilage. I also felt the extensor tendons gathered much together on the inner part of the cavity;



Torsion of the Astragalus (90°). (The bone should have been represented as being a little more abducted.)

they assumed their usual places when the foot was turned into its normal position. The anterior tibial artery was beating well.

The foot was placed on a T-splint and slung. The man passed a fairly comfortable night, expressing himself as much relieved by the operation. On the fourth day the temperature rose to 102·4°; after dressing the wound and the action of a laxative it sank to 100·3°, and since that time remained normal, except on one night, when it rose to 100°.

The skin wound healed mostly by first intention, save a little part anteriorly which gave passage to a small amount of pus formed in some part of the deep cavity. Therefore on December 17th I made a counter opening in a more dependent part below the outer malleolus through the recent scar.

January 13th.—I found the union between the leg and foot affording good resistance. All wounds have been healed a week ago. Limb put in plaster of Paris. To get up.

24th.—Discharged nine and a half weeks from date of operation.

Several points present themselves for consideration in this and in the very few similar cases recorded. Firstly, as to name. Recent writers have endeavoured to avoid the confusions of an older terminology by naming luxations between the astragalus and leg bones "dislocation of the ankle-joint;" such as take place between the former bone and the rest of the tarsus "subastragaloid;" while the combination of these two injuries is termed "dislocations of the astragalus.[1] But the word dislocation can hardly be with propriety applied to the relations between the leg bones and astragalus which subsisted in this case. Such a term would certainly tend to mislead, as denoting dislodgment or extrusion of the astragalus from the

[1] Boyer, 'Traité des Maladies Chirurgicales,' t. iv, p. 382, named these "double." The term "complete" is inadmissible, since it is already employed to mean the reverse of a partial dislocation—of a subluxation.

tibio-peroneal socket. I would propose to name these kinds of injury "Dislocations of the foot with version, and with torsion, of the astragalus."[1]

For it must be remarked that the bone may be turned within the tibio-peroneal socket either horizontally so that its long axis looks across the joint and at right angles to its normal position: this I would call "version." Or it may turn over sideways upon its antero-posterior axis: this might be named "torsion."[2]

Of the former injury I find by a very diligent search in all the records to which I have access, seven cases,[3] of which two are merely anatomical, being found in subjects examined for other causes. In all these cases save one the front of the bone was thrown inwards. In one case the head was broken off. In the four (inward version without fracture) the head presented below and sometimes also a little behind the inner malleolus; it appears to have a great tendency to become entangled between the tendon of the tibialis posticus and of the flexor longus digitorum muscles. In only one case did version occur outwards,[4] the turn was only a quarter of the circle (45°), the head of the bone resting against and in front of the external malleolus. It is, indeed, evident that the lower position and greater length of that outer projection would prevent the head of the astragalus passing under it as it does on the inner side, therefore, the twist being checked, a mere partial version in this direction can occur.

Torsion would at first sight seem a more common injury, for Malgaigne reports no less than six cases;[5] but on

[1] Malgaigne, 'Traité des Fractures et des Dislocations,' terms these "Luxations par Rotation sur place" and "Luxations par Renversement." Such words do not indicate the displacement at the tarsal joints of the astragalus.

[2] It would seem from the description of one case (Thierry) that the two twists may be combined.

[3] An appendix added to this communication gives the pathological positions and other particulars of each case.

[4] This was the analogue of a subluxation, and I cannot but think might have been reduced. (See Case 6 *b*, in Appendix.)

[5] Malgaigne also gives a plate, without history of this form of injury. The

referring to the originals it becomes plain that these numbers must be very considerably corrected, indeed, almost entirely rejected.[1] Malgaigne has fallen into one error by quoting the same case twice over from two different sources. Of the five remaining all, except one, must be rejected, because in four of them the astragalus, though certainly twisted, was also displaced out of the tibio-peroneal socket. In the one valid case the bone lay with its posterior end beneath the tibia. These statements can be verified by reference to the appendix, in which I have quoted the description of each case separately.

It appears that the distinguished author was misled, partly by an imperfect acquaintance with English in his interpretation of very unskilfully worded descriptions; partly by haste in accepting such words as "turned" or "*mouvement de rotation*" as signifying the condition in question, without going further to ascertain whether change of place did or did not accompany change of position.

Nevertheless Malgaigne's plate from the *Musée Dupuytren* represents an astragalus slightly displaced forward and turned on its long axis one eighth of the circle; its inner trochlear edge resting on the horizontal articular facet of the tibia. Thus, although my case is the only one recorded in which the four sides have each assumed the position of another while the bone remained in its socket beneath the tibia, yet it is evident that such accidents may, indeed probably have, taken place, and therefore are worthy of careful study and intelligible description.

The light which my case throws upon the subject appears to render the mode in which these injuries occur comprehensible. Two factors appear to me indispensible:—First, that during the time that the foot is twisted it should be at a right angle to the leg; secondly, that there be a cer-

astragalus is represented as partially turned, the inner edge of the trochlea looking upwards.

1 Correction seems the more necessary, since the writer of the article, "Dislocation," in the new edition of 'Cooper's Surgical Dictionary,' has in all faith accepted the account given by Malgaigne.

tain relationship of strength between the ligaments of the ankle and the calcaneo-astragaloid or interosseous ligament. When the gig my patient was driving fell over towards the right, the sole of his foot resting on its floor was made to face more and more inward until, no doubt, the external lateral and the outer parts of the interosseous ligaments were unusually tight. At that moment the outer border of the foot, then looking almost directly downward, came in contact with the ground, and thus was completed, by the impetus of a blow, what a more gradual force could not probably have effected. Had the foot been extended or flexed, as well as inverted, the astragalus could surely not have escaped dislocation backwards or forwards and outwards. Or had the interosseous ligament been weak in proportion to the lateral ligaments of the ankle, subastragaloid dislocation alone would undoubtedly have been produced. From this, too, we may infer that version results when the foot, while at right angles to the leg, is forcibly turned horizontally inwards (inverted), and is used as a lever wherewith to wrench round the astragalus in the intermalleolar mortice. Strength of the interosseous ligament is here also a necessary constituent.

My case, the only one I believe in which diagnosis has been arrived at from a study of the symptoms during life,[1] shows that certain signs which may be taken as pathognomonic can by care in examination be made out. Thus, since the astragalus was clearly absent from its normal place between the os calcis and the scaphoid, and since the head of the bone lay in front of the *outer* malleolus, the question as to the position of the body of the bone required solution. Now in all cases of dislocation of the astragalus, properly so called, the bone can be found somewhere about the foot or behind the bones of the leg, hence

[1] Malgaigne, and following him other writers besides those named in the text, describe the diagnosis of this condition as impossible. That opinion is, I believe, much too hasty; it appears to me that in the absence of any considerable swelling the diagnosis founded on anatomical knowledge can in most cases be made.

the conclusion that the bone was still in the socket was inevitable. But the abnormal position of the head showed that some change in its relations to the intermalleolar socket had taken place, that is to say, that the case was not merely a subastragaloid dislocation. The key to the problem was furnished by the bony ridge described in the history of the case as running back from near the head of the astragalus to the angle between the outer malleolus and the anterior edge of tibia. In a normal foot fully extended, the outer edge of the trochlea can be felt in precisely that situation: but when the position of the bone is so changed that its head lies in front of the outer malleolus, that edge of the pulley must also have moved away, therefore the ridge which I felt must evidently be some other part of the bone, and as there was no fracture, a twist of the astragalus must of necessity have occurred. The peculiar shape of that ridge, and the fact that it was convex *outward* instead of *upward* and forward, led me to conclude that the trochlea lay against the outer malleolus.

The diagnosis of version is, I should judge, still more easy; for, in all cases uncomplicated by fracture, the head of the bone could easily be felt; in one in front of, and a little below the outer malleolus; in five below, and a little behind the inner malleolus. In one case in which the head was broken off, the trochlea was visible through a wound under the inner malleolus.

Concerning treatment very few words are necessary. Any unreduced dislocation of the astragalus, save in the backward direction, very generally ends in sloughing of the integuments and wide-spread suppuration, unless the bone be removed. Of the six instances of "version" collected in the appendix, one is an old injury and does not seem to have been followed by suppuration, but the man must have been very lame. The cases of delay and hesitation all ended badly.

One case of "torsion," since it is an old injury, viz. that depicted by Malgaigne, does not appear to have been

productive of any acute troubles, but the displacement was only slight. Only one other case of veritable "torsion" (Case 4, p. 53, in Appendix) is on record; in that case the bone was almost entirely extruded from the tibio-peroneal socket and was excised. In my case it was impossible to replace the foot until the astragalus was removed.

A certain difference of opinion has existed as to whether it is better to excise at once or to wait until inflammation and suppuration have loosened the connections of the bone. Probably at the present day it is hardly worth while to refute this latter contention. My view is that in all cases of dislocation of the astragalus certain and sufficient, but not too persevering, attempts at reduction should be made. If they fail, the bone, unless it be luxated backward, should be at once excised.

Both version and torsion when complete, that is to say when the twist amounts to 90°, are irreducible. In the former case, excision is the best practice, though it is not so imperative as in the latter. When torsion is complete, the only choice lies between an utterly useless foot, excision of the astragalus, and amputation.

It is well known that excision of the astragalus leaves a very useful foot, as may be seen by the preparation on the table taken from Charing Cross Hospital Museum (a patient of Mr. Canton's), and by the way the subject of this paper, now in attendance, walks.

Postscript.—At the end of April, 1883, George F—, presented himself at the hospital walking with a barely perceptible limp. He said that if he had to go far his ankle got a little tired, but that this and occasional pain on change of weather were the only remains of the injury. He wore a shoe about one third of an inch thicker than that on the other foot.

APPENDIX.

Abstract of cases previously recorded.

Version.

1. *Laumonier* ('Journal de Fourcroy,' 1791, t. ii, p. 60). The head of the astragalus protruded under the inner malleolus, between the tendons of the tibialis posticus and flexor longus digitorum, the trochlea lying transversely in the socket and forcing apart the tibia and fibula. The bone excised with success ('Revue Med.-Chir.,' 1855, p. 207).

2. *Denonvilliers.*—The head was broken off and remained attached to the scaphoid, the body of the bone turned at a right angle; there was a wound eight centimètres (2½ inches) long, exposing the inner malleolus; below and a little behind which the trochlea was visible. "The projection of the tendo Achillis has disappeared, as also that of the heel, the whole foot seems to have moved bodily forwards, its inner border and lower surface are quite insensible, while the wound itself is extremely painful." On the second day exsection of body of bone; gangrenous inflammation; death on sixth day.

3. *Foucher* (a dissecting-room subject).—The astragalus had turned in its socket, the head had left the scaphoid and was under the inner malleolus, to which it was attached by firm fibrous adhesions, so that before dissection it was mistaken for an abnormally prolonged inner malleolus. Other changes were observed but the injury was evidently very old.

4. *Thierry.*—The head projected about midway between the internal malleolus and the tendo Achillis; the front part of the trochlea (*la gorge de la poulie astragalienne*)

lay just beneath the malleolus, while the posterior border of the bone was directed forwards and outwards, being placed between the calcaneum, cuboid, and scaphoid. "Thus the bone had turned on two axes; on its vertical, so as to place the head behind the inner malleolus, and also on its longitudinal, so that the trochlea became vertical and looked inward and forward; while the outer border lay below the inner above and the lower face looks towards the back part of the outer malleolus." Thierry temporised; sloughing occurred, through which the position above described was seen; excision; persistence of inflammation; amputation; recovery.[1]

5. *Verebely* (of Pesth).—Vigorous man, æt. 29. Tibia uninjured; fibula fractured above the malleolus. Under the internal malleolus the skin was very tense; about an inch lower there was a hard and bony enlargement about half an inch in diameter. Reduction failed. At the end of the third week an abscess was widely opened and showed that the enlargement under the inner malleolus was the head of the astragalus turned round to that situation. After four months' treatment the man could with difficulty put the foot to the ground.

6. *Verebely*.—Man, æt. 45. The foot was at right angles with the leg, the sole looking somewhat inwards and upwards (*etwas nach innen und oben*). "Under the easily distinguishable outer malleolus and in front of it a bony projection half an inch in diameter may be plainly felt; this can be moved without much pain backwards and forwards independently of the other bones. Behind the navicular is a considerable hollow." Attempts at reduction failed ('Wiener Medizinische Wochenschrift,' 1869, Nos. 17 and 18).

This case, referred to in my text as the only one in which version had taken place outwards, is compared to a subluxation, and for anatomical reasons we must conclude it to have been reducible.

[1] All the above cases are in the 'Revue Medico-Chirurgicale,' 1845, p. 203, reported by Foucher.

TORSION.

1. *Boyer.*—Of very doubtful reality. The description may be given in his own words ('Maladies Chirurgicales,' vol. iii, p. 899). "Un homme de 36 ans d'une petite stature, mais fort et vigoureux, tombe de cheval; son pied reste dans l'étrier pendant que le cheval continue de galoper; l'astragale éprouve la double dislocation dont il est ici question." (Boyer is writing of what is now called dislocation of the astragalus). "Cet os etait renversé sur le tibia, et sa tête, qui avait abandonné la cavité scaphoide en sortant par la partie interne supérieure de la cavité de cet os, formait sous la peau une tumeur très remarquable." In eighteen days the skin over the head sloughed, and the limb was amputated. Boyer only says of the condition, "L'examen anatomique de la partie fit voir la double luxation de l'astragale et les surfaces articulaires de cet os cariées, ainsi que celles du tibia et du peronée." Surely if he had seen the bone within the tibio-peroneal socket he would have made some remark on so singular, and at that time unknown, a condition, nor would he have termed it double dislocation.

2. *Smith* of Leeds (Turner on 'Dislocations of the Astragalus,' p. 69).—Man, æt 66. "There was a projecting part of the bone in the situation of the head of the fibula and a little lower another portion of bone; these were covered by the common integuments, much upon the stretch, the bones were firm and immovable." In a week the integuments had sloughed, "it was now distinctly seen that these two points which projected were portions of the same bone." Mr. Smith immediately suspected it to be the astragalus which was dislocated, and upon "comparing it with one of those bones the upper portion was found to be the outer surface of the bone which articulates with the tibia, and the lower portion was found to be the head of the astragalus which articulates with the scaphoid bone." Eighteen days afterwards the bone was excised

and the man made a good recovery. It seems impossible to assume here that the bone was in the socket, since the trochlea or the outer surface presented at the wound. The description is very obscure.

3. The preparation which Malgaigne says Liston possessed is thus described ('Pathological Transactions,' vol. i, p. 45): "*Dislocation of the Astragalus* which had become separated from its connections with the os calcis and the bones of the leg, neither the tibia nor fibula having been fractured or separated from each other. The astragalus was so tilted on itself that the under surface looked towards the outer malleolus, being confined by the peronei tendons, which passed over its posterior aspect; the head was so tightly embraced by the annular ligament and tendons of the extensor longus digitorum as to render reduction impossible. An analagous case had come some time before under the notice of Mr. Liston in which, although the foot was put straight, it was impossible to reduce the bone—5th April, 1847." I confess myself unable to interpret this description—it is clear that the bone had turned, but the first sentence would point to real dislocation; to extrusion from the tibio-peroneal cavity.

4. *Dupuytren's* first case (according to Malgaigne) is to be found in the old Sydenham Society's publications, 1847. (Dupuytren 'On Injuries and Diseases of Bone,' translated by Le Gros Clark: also in Rognetta's 'Memoir on Extirpation of the Astragalus,' p. 29.) M. G—, æt. 50, coach-builder, jumped from a ladder and alighted on the heel. "The medical man first called in, found a large, hard, irregular, and irreducible prominence in front of the tibia and extending to the instep." "When consulted, Dupuytren at once was satisfied that this was the astragalus." Attempts at reduction being useless, an incision was made through the skin parallel to the axis of the foot, and the head and neck of the astragalus were immediately brought into view. These were seized, but efforts to remove the bone were vain; for the posterior part was grasped and held fast between the tibia and os calcis.

Surprised at this unexpected resistance we attentively sought for the cause and discovered not without extreme astonishment that the astragalus was turned round in such a way that its upper surface was directed downwards, its lower upwards, and that the hook-like process at its inferior and posterior part was fixed beneath the tibia so as to completely frustrate our efforts to extract it" (the patient ultimately did well).

From this description it is plain that the interosseous groove of the astragalus was caught against the anterior edge of the tibia, therefore, that nearly the whole of the bone was extruded from the socket.

Dupuytren's second case ('Leçons Orales,' vol. ii, p. 18, also quoted by Rognetta p. 31, and referred to by Malgaigne) "L'astragale avait eprouvé un mouvement de version tel que la face supérieure etait en dehors et devenue externe, c'etait elle qui se marquait sous la peau ainsi que la face externe qui etait devenue supérieure . . . Sous la peau de la face dorsale du pied ou plutôt sous un cicatrice inégale ondulée, l'astragale saillant donnait en quelque sorte au membre la difformité d'un pied bot."

From this description it appears that the astragalus was extruded from its socket, and was not the sort of case which Malgaigne imagined and named a "rotation sur place," or the astragalus would not have been thrust forward on the dorsum of the foot. These cases are the only ones of Dupuytren's mentioned by Rognetta which bear the slightest resemblance to the injury in question; therefore, Malgaigne, in ascribing to the former surgeon three cases, has quoted one or the other twice over—for Rognetta's quotations are from the same sources that Malgaigne had previously named.

The figure in Malgaigne's atlas has no history attached—it is from Dupuytren's museum—it is an old injury, since anchylosis had taken place. The torsion is very slight, the outer angle of the trochlea rests against the inner surface of the tibia (barely 45°). The body of the bone is slightly misplaced forward.

ON

RESECTION OF PORTIONS OF INTESTINE.

BY

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(Received October 5th—Read December 12th, 1882.)

THE operation of resection of certain portions of the bowel for various diseased conditions has now been performed so many times and with such a proportion of success that surgeons are in a position, I think, to consider not only the justifiability of the procedure, but also the circumstances that best conduce to its successful issue.

Indeed, from an examination of the cases already published, it would appear that the operation is not only justifiable, but that in selected and proper instances it is the best and perhaps the only means of affording substantial relief or reliable cure. Resection has now been practised upon all parts of the intestinal canal from the pylorus to the rectum, but I will confine my remarks to the principal difficulties and risks of the operation, and endeavour to point out how those difficulties may be met, concluding with an account of a case in which I recently resected a portion of the descending colon by a new method. A careful examination of the recorded cases of resection, and

especially of the fatal cases, reveals one very striking fact, and that is that the failure of the operation is more often due to defects in technical details than to any other cause. The future of the operation, and the probability of its being established as a proper and sound surgical procedure, depend, I think, at the present moment, more upon improvement in the details of the operation itself than upon a more elaborate knowledge of the circumstances under which it should be performed.

In discussing the items of the operation I will consider first resection of the small gut, and subsequently resection of the colon, omitting altogether the operations for pyloric cancer and for artificial anus.

In resecting portions of the small intestine the abdomen is opened in the middle line below the umbilicus. When the peritoneum has been divided, and the diseased part is being searched for, some protusion of the distended gut is very usual. It is most desirable that this protrusion should be prevented. With care the intestines can be retained by the application of large carbolised sponges, and by puncturing the distended bowel in several places with a capillary trochar. This pricking I have practised without ill result in three cases of laparotomy for intestinal obstruction. When the diseased part has been dragged out of the wound, it becomes necessary to control the gut above and below to prevent extravasation of fæces when the resection is performed. This control of the bowel has been effected in many ways. By some it is advised that a partial union of the bowel should be effected before the preparations for an intestinal fistula are made. Of the other methods available there are Jobert's invagination process, which consists of invaginating the upper into the lower end of the completely divided intestine and then stitching together the serous surfaces, and Maisonneuve's plan of "intestinal anastomosis," where the divided small gut above the obstruction is joined to some part of the colon. Neither of these processes, however, would appear to have been applied to the human subject. There remains

the method of directly uniting the two ends by suture, returning them into the abdomen and closing the parietal wound. The difficulties in the way of this last and perhaps most desirable method are numerous. The two ends of the gut have to be kept in accurate apposition while the sutures are being inserted. The corresponding parts of the two portions of the bowel must be precisely approximated. Any sudden movement of the parts may cause the sutures already inserted to tear out, and the relation of the divided ends to be disturbed. Moreover the intestine operated upon is lax, flabby, collapsed, and empty, and the accurate adjustment of sutures to such a structure is a matter of considerable difficulty. A still further obstacle is found in the fact that the gut above the occlusion is usually much dilated, while that below it is narrow and shrunken. The two cut ends under these circumstances are so disproportionate in size that the union of the two by suture becomes a problem somewhat difficult to solve.

From what has been said it will be seen that the following circumstances may contribute to a failure of the operation. The bowels may be unduly exposed and handled during the operation. The gut may be damaged by the clamps or ligatures applied to prevent extravasation of its contents. The difficulty of introducing the proper number of sutures may render the operation of long duration, and on this account it has on more than one occasion occupied over two hours.

Fæcal extravasation may occur after the united gut has been returned, owing to the improper manner in which the stitches have been applied, or to their insufficient number. In one instance (Baum's case of resection of part of the ascending colon) only six sutures were applied, with the result of fæcal matter escaping soon after the operation was completed. When one remembers that in cases of resection of the pylorus from forty to sixty separate stitches are not considered too many, it will be obvious that six sutures applied to the colon will be

almost as useless as one. In several cases the fatal issue appears to have been due to a kinking or abrupt angular bending of the gut at the seat of the resection. This result can, I think, be avoided by taking care that the gut at the seat of the operation is well occupied by intestinal contents before it is returned into the abdomen. If moderately distended the gut has little opportunity for bending, while the distension serves to test the security of the stitches. In the second place, a proper treatment of the mesentery may help to prevent occlusion at the suture line. The mesentery attached to the piece to be resected should not be divided in a straight line close to its attached border as has been usually done. The better plan is to remove a triangular piece, the base of the triangle corresponding to the portion of bowel to be removed. The edges of the gap thus formed in the mesentery should then be carefully approximated by a few points of suture, and care should be taken to remove no more of the membrane than that corresponding to the segment of gut to be excised.

In two or three cases, from want of proper clamping, fæcal extravasation occurred from rupture of the gut during the necessary handling of the part, and a fatal issue was thus at once rendered almost inevitable. I have been unable to find any case of stricture of the gut following the direct suturing of the intestine after resection. In Rembard's case a piece of the suture used was found in a stool twelve days after the operation. It would appear that the sutures are soon covered over with lymph, and in a case reported by Berger this protection is said to have occurred eight hours after the operation.

That the integrity of the gut can be fully established after resection followed by suturing of the divided ends is, I imagine, proved by the cases of pyloric resection where the test is as severe as it can be. In nine experiments made upon animals by Madelung perfect union of the divided bowel occurred after the resection of a segment of the intestine.

As the operation of resection, however, at present stands, it must be confessed that the best results have been obtained in those cases where the excision has been followed by the formation of an artificial anus; and, on the other hand, little encouragement has been offered for the performance of direct apposition of the divided ends and the replacement of the bowel within the abdomen. This latter procedure has failed from gross faults in the details of the operation, and if those faults be remedied I see no reason why this method should not surpass in its completeness and in the certainty of its results the plan that involves the formation of a temporary or permanent intestinal fistula.

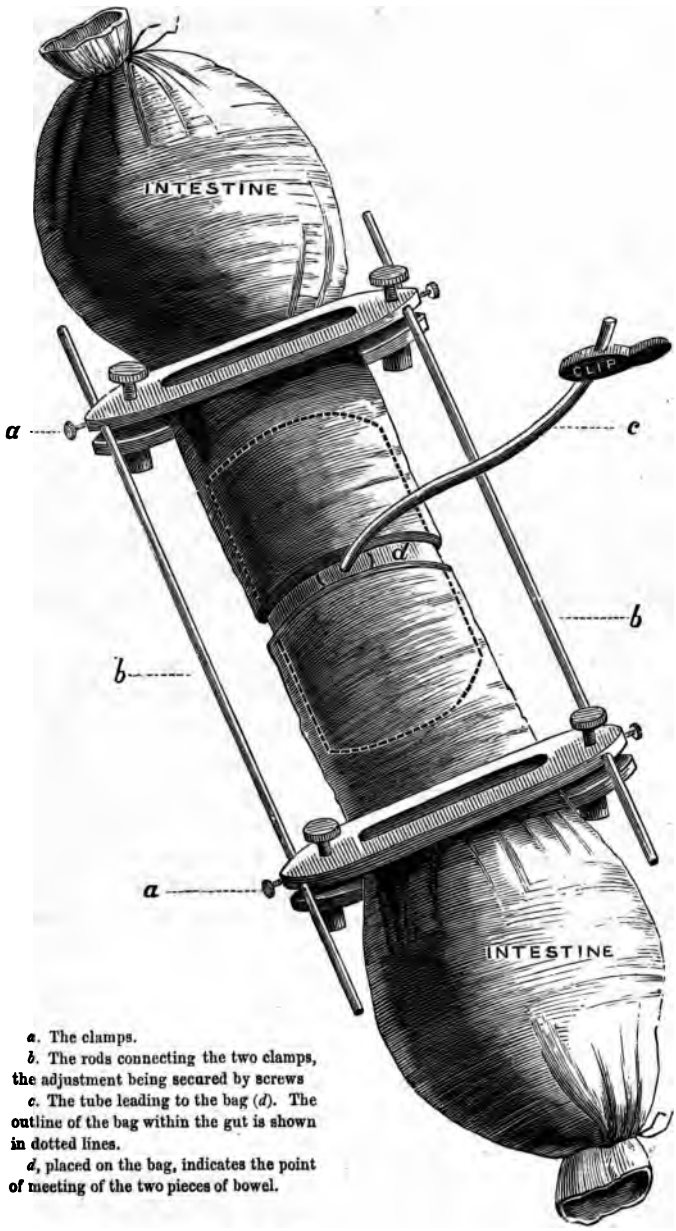
In regard to resection of the colon the above remarks will, with some modifications, fully apply. The great difficulty in resection operations performed on the large intestines lies in the situation of the abdominal incision. When a distinct tumour exists and when the precise locality of the disease is obvious, it may be the best practice to cut down upon the mass in one or other loin, making use of one of the incisions for lumbar colotomy. When, however, it is impossible to diagnose the site of the disease with absolute certainty, there appears to be no alternative but to make an exploratory incision in the middle line. This has been done in two cases (Gussenbauer's and Marshall's), while in a third case (Baum's) the incision was made two inches and a half to the right of the middle line. In Baum's case the disease was in the ascending colon, in the two other cases in the descending. In each of the three instances it was found impossible to remove the mass through the median incision. In Marshall's case, therefore, a second wound was made in the loin, and in the other cases a free incision was carried transversely to the loin from the original wound. It is significant that these three cases are those that were fatal out of the seven recorded cases of colectomy, although the gut was in each instance removed finally from the lumbar region as in the non-fatal instances.

There are many points in favour of resecting the colon from a median incision, and this applies even to cases where a mass is obvious. That it can be easily done, if proper appliances are used, is, I think, demonstrated by the case given below in which I resected without difficulty a portion of the descending colon through a wound in the middle line.

In the lumbar incision the space is limited and it has been found difficult to fully expose a large mass in that locality. In one case (Martini's) it was found to be impossible to reunite the colon after the resection, and thus a permanent artificial anus was inevitable. . This difficulty would not have been encountered had the bowel been approached from the middle line. Of course if it is intended that an artificial anus shall be formed after the resection, the loin is the proper spot to select for the operation. But if it is intended to resect the bowel and then to unite the divided ends by suture, I am strongly of opinion that the median incision is the best. In the discussion that followed upon Mr. Bryant's paper read before this Society in March last, I find it reported that two surgeons, Mr. Howard Marsh and Mr. Harrison Cripps, were inclined to advise if not a median incision at least a vertical incision at the outer edge of the rectus. They contended, with I think considerable justness, that the incision in the loin did not afford sufficient room for other than very limited operations.

I will now beg to draw the attention of the Society to an appliance I have had made that will, I hope, meet many of the difficulties incident to resection.

The apparatus consists in the first place of two clamps to secure the gut above and below the site of resection. Each clamp is made of two separate and light metal bars, provided with an india-rubber pad on the surfaces that are in contact with the gut. The clamp is two inches and a half in length, and one part being placed beneath the gut and the other upon it, the two are then approximated by screws placed at each end. By this



a. The clamps.
b. The rods connecting the two clamps, the adjustment being secured by screws
c. The tube leading to the bag (*d*). The outline of the bag within the gut is shown in dotted lines.
d, placed on the bag, indicates the point of meeting of the two pieces of bowel.

means the gut can be evenly and accurately compressed with as much or as little force as may be thought fit. I find that a clamp of the size exhibited is equally suitable to the small gut of a child and the distended colon of an adult. I first apply a clamp to the gut one inch and a half below the proposed resection line and then having emptied the part to be excised by squeezing its contents upwards, I apply the second clamp at a similar distance beyond the upper resection line. Thus in most cases an empty condition of the part to be removed can be insured. A triangular piece of the mesentery should then be excised, the base of the triangle exactly corresponding to the part of gut to be removed. All its divided vessels having been secured the diseased gut can be at once resected. I now unite the corresponding ends of the two clamps by means of long narrow steel rods secured to the extremities of each clamp by a small screw. By means of these bars the two clamps can be evenly approximated and the divided ends of the gut brought into the most accurate contact.

When all the screws are secured the bowel to be united by ligature is held in a rigid frame, so that it can be turned over or moved in any direction without disturbing the contact of the two divided ends. By this means the sutures can be as readily applied to the sides and under surface of the bowel as to its upper or more exposed surface. The frame does not require to be held and any risk from the slipping of the divided intestine is quite avoided. The sutures may then be introduced. Now, although the frame maintains the bowel ends in accurate and stable position, yet those portions of gut, being limp and collapsed, are therefore not easily united. What appeared to me to be required was some solid tube or support that could be introduced at an equal distance into the two ends of the bowel and over which the sutures could be applied. For this purpose I devised a tube of fine gelatin about one inch in length proposing to insert equal portions of it into the two bowel ends and suture

the parts over it as over a substantial support. I imagined that the gelatin tube would after the operation prevent kinking of the gut, would lessen the risk of extravasation of the contents and would keep the tube patent. Moreover, it would possibly not be dissolved until the sutures had been rendered somewhat more secure by lymph. But to be of any use these tubes would have to be kept in a multitude of sizes, and moreover they would hardly be of any use under those common circumstances where a distended bowel above an obstruction has to be united to a shrunken bowel below it. Discarding these tubes I had a very thin india-rubber bag made about three inches in length, of sausage shape, and that could be distended by air to a large size through a tube inserted about the middle of its long axis. Having blown out this bag till it was about the size of the divided bowel, I inserted one end into the lower piece of the intestine, and the other end into the upper piece. The tube that supplied it with air would thus occupy the suture line. After being inserted the bag can be distended to any size, and can thus quite overcome any inequalities in calibre that may exist between the two pieces of bowel. It moreover forms a firm support in the interior of the gut over which the sutures can be most readily applied. If the distension of the bag disturbs the mutual relations of the divided ends, this can be rectified by a slight readjustment of the clamp. The sutures are then applied all round the gut and almost close up to the interruption in the suture line caused by the tube that fills the bag. The sutures having been applied, the bag is exhausted of air and is withdrawn from the bowel, and so thin is its structure that the collapsed bag can be drawn through a hole with the circumference of a No. 13 catheter. It only remains, then, to close the little opening left after the removal of the bag. If the stitches are properly applied, there should not be the least risk of pricking the bag, for the sutures should not include the mucous membrane. Of the various forms of suture, viz. Gely's, Czerny's, Güssenbauer's, and Lem-

bert's, I think that the last named is to be preferred as it is the most easy to introduce, and in this operation I imagine that time is a matter of great moment. At least from thirteen to twenty points of suture should be employed. Madelung advises that they should not be more than three millimètres apart. The best material for the stitch is, I believe, the finest "Chinese twist," and in using Lembert's suture I have found a small curved needle used with a needle holder the most convenient. Before the gut is replaced in the abdomen and the wound in the parietes closed, it is necessary that the two cut edges of the mesentery be united by a few points of suture.

I will conclude this paper by very briefly detailing a case of resection of a part of the descending colon. This is the third time, I believe, that the operation has been performed in this country, and it is, so far as I am aware, the only instance on record where a portion of the colon was excised through an incision in the middle line. The patient I might state was in a very prostrate condition at the time of the operation and died in twelve hours.

The patient, a sailor, æt. 56, was admitted into the London Hospital under the care of my colleague, Dr. Warner, on September 1st. 1882. There was nothing of note in the details of his previous history. For two months before admission he had suffered from occasional attacks of colic associated with slight vomiting and absolute constipation, and then followed by diarrhœa. On admission he was feeble and was losing flesh, his face was contracted and his eyes sunken. He could take but little food and vomited a great deal of what he did take. His bowels were as a rule constipated, but were relieved occasionally by a species of diarrhœa. He complained of repeated attacks of severe abdominal pain associated with movement of the bowel that could be felt by the hand. The abdomen was moderately and evenly distended; there was no abdominal tumour and nothing to be felt per rectum. On an enema being given 4½ pints were easily introduced before any returned. These symptoms continued with

some modifications until September 12th; the man in the mean time becoming feebler and more wasted. On this date he became much worse, the vomiting was severe and continuous, the pain constant, and the abdomen more distended. When I saw him on September 14th, he was extremely feeble and much exhausted, and troubled by an almost constant vomiting that, however, was never stercoraceous. I had ventured to diagnose a stricture of the bowel but could find no indications as to its probable site. At 10 p.m. the man was anæsthetised and the abdomen once more very carefully examined with the same negative results. I then made an incision in the middle line below the umbilicus. The first part of the intestine to present was a greatly distended cæcum. Introducing the hand into the abdomen I proceeded to examine the whole length of the colon and soon found a small hard mass in the descending colon about two inches or so below the splenic flexure. To fully expose it, the small intestines were allowed to protrude. They were deeply congested and presented a vast number of blood-extravasations under their peritoneal covering. They were carefully protected by warm carbolised cloths but I feel sure that to allow the escape of these parts was a mistake in the procedure that may, and should have been avoided.

With a blunt director I divided the peritoneum that was reflected from the descending colon to the parietes and with some little manipulation dragged the strictured bowel towards the middle line. I soon had freed it sufficiently to allow of one clamp being applied above the seat of disease, the other clamp having been already applied to the gut below. I divided the peritoneal connection of the part to be removed, resected it and applied the sutures to the divided ends of the bowel in the manner already described. The frame answered admirably and without its use I believe that the resection would have been impossible owing to the depth at which one had to work. The clamps, frame, and distending bag were all employed in this case. I introduced twenty-five sutures after Lembert's method, bringing

the two serous surfaces together and avoiding the mucous membrane with the needle. The clamps were then removed, and the fæcal matter in the distended gut above the resection line was squeezed down so as to occupy the part just operated upon. I was glad to find that even when pressure was applied to the part above no escape from the gut of its contents occurred at the line of the sutures. The bowel was replaced and the abdominal wound closed. The operation took one hour and a quarter and was performed under strict antiseptic precautions.

At the autopsy there was commencing general peritonitis. There was no trace of blood in the peritoneal cavity and not the least escape of fæcal matter at the site of the operation. The colon at the line of the sutures was distended with fluid fæces so that it was as large as the engorged bowel above it. A quantity of fæcal matter had likewise found its way into the rectum. The stricture was composed of a dense mass about the size of a walnut. It surrounded the bowel equally at all parts, and had so narrowed its lumen that the tube would barely admit the tip of the little finger. It proved on examination to be a cylindrical epithelioma.

It is much to be regretted that the operation was not performed sooner, for the patient was in so feeble a condition when anæsthetised that his recovery from even a milder operation would have been a matter of question. It would possibly have been better if in this patient I had adopted the excellent practice carried out by Mr. Pitts in a like case.[1] Mr. Pitts—in a case of stricture of the descending colon, that could not be diagnosed before the abdomen was opened—made a median incision, discovered the mass and then performed colotomy in the left loin, proposing to excise the mass at a future period. His patient made a good recovery. It is extremely doubtful, however, if my patient could have borne this double operation.

The case—so far as it goes—appears to demonstrate

[1] 'St. Thomas's Hospital Reports,' vol. ii, 1882, p. 75.

that with proper appliances a portion of the colon can be resected through a median incision, and that the gut can be so united after the excision of a portion as to quite prevent any escape of the intestinal contents.

A CASE

OF

ANEURISM OF THE EXTERNAL CAROTID ARTERY;

LIGATURE OF THE COMMON CAROTID, WITH CURE OF THE ANEURISM. DEATH FROM PARALYSIS ON THE THIRTY-FIFTH DAY.

BY

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(Received November 27th, 1882—Read February 13th, 1883.)

In the sixty-fourth volume of the 'Medico-Chirurgical Transactions,' Mr. Henry Morris has recorded a very interesting case of aneurism of the external carotid artery, in which, after failure of ligature of the common carotid to effect a cure, the old operation of incising the sac was performed, and ligatures were placed on the facial and lingual arteries and upon the main trunk of the external carotid above the sac, with ultimate recovery. The conclusion which Mr. Morris draws from his case is that "it would be best, in carotid aneurisms near the bifurcation of the common trunk, to ligature simultaneously the common carotid and such branches of the external carotid as are easily accessible, viz. the facial, superior thyroid, and lingual, which may all be reached through one oblique incision, and the temporal." In the discussion which followed the reading of Mr. Morris's paper on 26th October, 1880,

I ventured to dispute the correctness of this statement, and I bring forward the following case in support of the view that ligature of the common carotid alone is sufficient to cure some cases of aneurism of the external carotid.

As regards the position of the aneurism in the following case the diagnosis was much assisted by the fact that the right half of the patient's tongue was considerably atrophied, presumably from pressure by the aneurism on the hypo-glossal nerve where it crosses the artery. It seemed unlikely that the internal carotid could, even if aneurismal, sufficiently stretch the nerve to cause paralysis, and there was no reason to think that the bifurcation of the common carotid was higher than usual. The occurrence of aneurism at the age of twenty-three was sufficiently accounted for by the state of the valves of the heart and the liability for embolism to occur in consequence of detachment of a vegetation. Death was clearly due to embolism through the *left* carotid from this cause, and in no way depended upon the operation, from which the patient had completely recovered, the aneurism being solidified throughout. A carbolised silk ligature was used with antiseptic dressing, and appears to have undergone no change.

For the very carefully recorded notes of the case I have to thank Mr. Stanley Boyd, late surgical registrar to University College Hospital.

Annie K—, æt. 23, a tailoress, was admitted into University College Hospital under the care of Mr. Heath on January 7th, 1882, with an aneurism of the external carotid. She gave the following history:

On December 10th, 1881 (one month prior to admission), she came down in the morning talking indistinctly; on her mother remarking this she looked at her tongue and found that it deviated to the right, even more markedly than on admission. On the same day she felt something wrong with her throat, not soreness, but chiefly difficulty in swallowing. Her neck was rubbed with oil for four days, when (December 14th) a swelling as large as a

filbert was found near the right angle of the jaw. She then went as out-patient to a hospital, where nothing was done for a week; but as the swelling continued to increase in size it was painted with iodine, and this treatment was carried out until the patient was admitted to University College Hospital.

When seven years old the patient had an attack of rheumatism, and since then she had had several; during the last twelve months she had scarcely been free from rheumatic pains, and her joints, especially knees, swelled frequently. She had been losing appetite, flesh, and colour for six or eight months, and sweating much at night, but had had no cough; vomiting had been frequent for some weeks past, and for some time she had been living on fluids, being unable to swallow solids.

On admission the patient was very thin, weak, and markedly anæmic. There was a smooth, round, pulsating swelling just below the right mastoid process, reaching down to about the level of the upper border of the thyroid cartilage, limited in front by the ramus and angle of the jaw, and overlapped behind by the sterno-mastoid, but no parotid could be felt on its surface. Altogether it had the size and shape of half a small orange, was very tense, and its pulsations were expansile. The right tonsil was pushed somewhat inwards, and pulsation could be felt beneath, but it was not strong. The right temporal pulse was markedly weaker than the left. The tongue deviated much to the right, and the right half of the organ was a good deal wasted. There was more dysphagia than the slight encroachment of the aneurism on the throat would account for; only fluids could be swallowed, and that with difficulty and pain. No dyspnœa was present.

There was no pain about the aneurism, but the patient complained that the pulsations shook her head. She said that her sight was often hazy, that she could not read for any length of time, and that these symptoms were more marked in the right than in the left eye. The pupils were normal, the optic discs were very pale, and the margins

obscure, especially the right. She also complained of occasional diplopia, but no squint was ever noticed.

The heart's impulse was strong, heaving, extending far outside the nipple line. There was no thrill. At the apex a loud, well-conducted, mitral, systolic murmur was heard; at the base a much fainter double aortic, a distinct second sound intervening between the murmurs. Pulse 100, full and strong. There was a little albumen in the urine. Temperature often 100°—102°.

On January 11th Mr. Heath tied the common carotid just above the omo-hyoid with a thoroughly carbolised silk ligature. The right pupil dilated somewhat immediately after the ligature was tightened, but the knot did not completely stop pulsation in the aneurism. The ends of the silk were cut off close, the wound was washed out with carbolic lotion, and closed by four wire sutures. No spray was used, otherwise the operation was done antiseptically. A gutta-percha splint was bandaged on to the head and shoulders to keep the face turned towards the left.

The patient slept well after the operation. On the second day the pupils were of moderate size, equal and reacting readily. A distinct difference in colour and temperature of the cheeks was noticed, the right being the cooler and paler, and this persisted till the third day. It was exaggerated by the left cheek being kept in constant contact with the pillow. The tongue deviated as before the operation, and no change was noted in it before death. The patient complained of rather more pain and difficulty in swallowing, but this soon passed off and power of deglutition steadily improved. The urine was rather dark from carbolic. The temperature was 101·2° on the morning of day of operation, 101° in evening; but on the second day it had fallen to 99·6°, and reached 98·2° in the evening.

On the third day everything was going on well. The dressing was changed under the spray; the edges of the wound were in perfect apposition, there was no redness

tenderness, or swelling. There was distinct pulsation in the tumour; the facial pulse was plain, but the temporal could not be felt. Temp. 100·2°—101°.

On the fourth day the patient vomited once; her urine was deeply carbolised, and there was one quarter or one fifth albumen in it. The albumen did not diminish in quantity up to the twentieth day, after which there is no note. The bowels were much confined and scybala had to be cleared out of the rectum by finger on the seventh day. At the dressing on this day (seventh) the wound was found to have united completely by first intention; the sutures were causing no irritation, so they were left in beneath an antiseptic dressing, which was renewed on the tenth and thirteenth days. On the seventh day it was doubtful whether any pulsation existed in the aneurism; on the tenth day it was certainly absent; and on the eighteenth the sac was smaller and quite hard. On the twenty-fourth day the right tonsil had fallen back to its normal position; and on this day the superficial temporal pulse was felt for the first time. On the twenty-fifth day the patient got up for a short time; and on the twenty-ninth the splint fixing her head was removed. Her power of swallowing had increased so much that on the ninth day she began taking chicken for dinner; but she vomited almost daily; her temperature was very irregular, being sometimes normal for two or three days and then 100°—101°—or a little over—for two or three more. The most marked attack of fever during this period was probably due to a subacute cellulitis of the right hand and forearm, which appeared on the fourteenth day and disappeared under fomentations in four days.

The patient continued to get up daily for two or three days; vomiting then became more severe and she was confined to bed. The patient felt pretty well, and continued to take plenty of food, but the vomiting was unchecked by all the remedies tried. Still it was not very severe, coming on only three or four times a day; and the patient did not seem to be losing strength. The

temperature was still irregular, often running up at night to 100°—101°, and once to 103·2°, whilst, in the morning it was generally 99° or a little over. There was no pain, but slight palpitation was occasionally complained of.

On the thirty-first day a plain aortic obstructive murmur was heard, whereas there was but a faint one on admission.

On the thirty-third day just after noon (12.15 p.m.) the patient was sitting up in bed, supported on pillows, eating some mutton. She suddenly dropped her knife and fork, and leaned back complaining of pain at the back of her neck and down the spine to the sacrum. She used both her hands to show where the pain was. After this she began to cry—a very unusual thing for her. She soon cheered up, but could not be persuaded to eat any dinner. At this time her speech was no worse than usual; but, at 2 p.m. it was unintelligible, though she evidently understood all that was said to her. At 4 p.m. she did not try to speak at all, and two hours later she did not recognise her friends, nor did she take any notice when spoken to, but lay quite still with her eyes partially open. Her temperature was 98·4°—101·6°. During the night swallowing became difficult, and finally, power of deglutition was lost; the motions were passed in bed. On the following morning (thirty-fourth day) it was found that the right arm was paralysed, and at 11 a.m. the right leg was similarly affected; but it seems certain that three and a half hours previously she had drawn this limb up whilst the bed was made. There was no ocular paralysis and no asymmetry of the face. No convulsions or twitchings had been noticed. The pupils were equal and reacted readily. The pulse was 99, full, compressible, thrill easily developed. Temperature 101·8°. It rose steadily to 105·2° at 7 p.m., and after this varied between 104·8° and 108·6° at which latter point it stood for three hours before death. This occurred at 5 p.m. on the thirty-fifth day. There was no post-mortem rise of temperature.

Post-mortem 19 hours after death. Temperature of room 53° F. Rigor mortis slight.

Brain: too much fluid on surface; a few small hæmorrhages here and there on the pia mater. No flattening of the convolutions; no difference in size or appearance of the hemispheres.

In the base of Broca's convolution was a vessel containing a small, firm clot, probably an embolum; but there was no surrounding hæmorrhage or anæmia. The vessel and clot formed a small patch about the size of a split pea. Five or six small patches of yellow softening were found beneath the grey matter of the surface; one beneath the right supramarginal convolution was the size of a threepenny piece, and another, rather larger, lay beneath the operculum, just external to the right claustrum—probably results of old emboli. Beyond this, and perhaps some diminution in consistence of the brain substance, the organ seemed normal. The arteries at the base appeared healthy but they were not slit up.

The body was thin and small petechiæ dotted the whole surface. There was some bronchitis, and collapse of the lower lobes of the *lungs*. *Pleuræ* normal. *Pericardium* normal. *Heart* much hypertrophied on left side. Blood well clotted, no staining of endocardium; right valves healthy. Mitral valves moderately thickened; many rather firm vegetations on both surfaces and borders. Each cusp of the aortic valve was studded, on the side towards the lumen of the vessel, by a button of vegetations, one being as large as a fair sized pea. The endocardium of the left auricle was also roughened over a large surface by small firm vegetations. The wall of the left ventricle was two thirds to three quarters of an inch thick, and decidedly too pale; one miliary abscess was found in it.

There were many petechiæ beneath the parietal peritoneum. *Liver* normal, but for a deep congestion of the hepatic veins. *Spleen* rather large and firm; many small and two large yellow infarcts in the surface, solid, slightly raised, friable: others, which had softened, in its substance. *Kidneys*: cortex narrow, pale, and firm; surface

deeply punctured from scarring of old infarcts? In the substance of the left were four or five softening infarcts; in right, one or two, and one on the surface. *Stomach:* many small submucous petechiæ; otherwise normal.

The scar of the wound through which the carotid had been tied was perfect. Immediately the platysma was reflected the sac of the aneurism, of a yellowish-brown colour, was exposed, surrounded by dense fibrous tissue which extended down to the bifurcation of the carotid. It formed a tolerably firm swelling, and on section, was full of red, laminated clot; the wall in parts was nearly one eighth of an inch thick. The shape of the aneurism was oval and its measurements were $1\frac{1}{4} \times \frac{3}{4} \times \frac{3}{4}$ inch. It sprang from the external carotid about half inch above the bifurcation of the main trunk, and the mouth of the sac seemed very wide, nearly half an inch of the wall of the vessel having yielded. The only vessel arising below the sac was the superior thyroid. The facial and lingual came off by a common trunk from its anterior part; they are closed at their origin, for a bristle cannot be introduced along the facial. From the upper and inner part rises the temporo-maxillary trunk. The origins of the other branches were not certainly made out.

The descendens noni ran up beneath the sac and was followed through dense fibrous tissue to its junction with the hypoglossal, which lay below and behind the sac, tightly stretched and of a yellow colour, either from fatty change or blood-staining. The aneurism had evidently risen from the external carotid just above the point of crossing the nerve, which, in its growth, it had pushed downwards and outwards. The sudden onset of the paralysis of the tongue would seem to show either sudden origin or sudden increase of size of the sac.

The ligature lay on the carotid rather above its middle point. Its site was marked only by a slight prominence over the knot, it was covered by loose connective-tissue, and there was absolutely no thickening or matting together of the soft parts about it.

On opening the artery above it a firm red clot was found, about three quarters of an inch long, adherent all round at its base to the walls of the vessel, but at its apex becoming more taper and adhering to the inner side of the vessel only. Below the ligature lay a small clot, not a quarter of an inch long, adherent only to the anterior side of the vessel.

A section through the scar in the skin, subcutaneous tissue, and platysma, enables one to recognise with certainty the line of the incision only in the skin; here, it is shown by a distinct depression on the surface, by the absence of papillæ, by indistinctness of the wavy bundles of connective tissue and by considerable increase in the number of nuclei; in many sections one or two considerable groups of small round cells are cut across, lying just where true skin and fat join. Beyond this point it is impossible to say from any disturbance in the arrangement of the bundles of connective tissue or of muscle, which direction the incision took; and, with the exception of very small collections of small round cells here and there, the appearance of the tissues is normal. It will be remembered that the wound healed by first intention, and that the patient died five weeks after its infliction.

It will be seen from the specimen shown (see Plate I), which has been carefully mounted by Mr. Shattock, that a piece of the ligature including the knot has been removed, but it was taken in such a way that no tissue was left inside the silk. Consequently, in most of the sections prepared, the silk has fallen out; here and there a bit has remained. On the outer side, the ligature is everywhere covered by a thick layer of loose connective tissue, free from signs of irritation, and often containing a few fat-cells close to the point through which instruments must have passed. As seen by a low power, the ligature site looks like the wall of a small abscess, so dense is the surrounding cell infiltration. With a high power it is found that a good deal of fibrous tissue has formed, constituting quite a capsule to the ligature at some points.

Young fibrous tissue, rich in cells, runs into the grooves of the knot, in some sections passing across the cavity between two pieces of silk. The bodies of many of the cells forming the infiltration are of considerable size, though they possess but one nucleus. Large multinucleated cells are by no means uncommon, in fact, all the signs of a chronic inflammation are present. Into the looser parts of the ligature round cells have penetrated some distance, into the tighter, scarcely at all. Fibrous tissue is forming in small quantities between silk fibres at some spots. There is no sign of erosion or absorption of the silk.

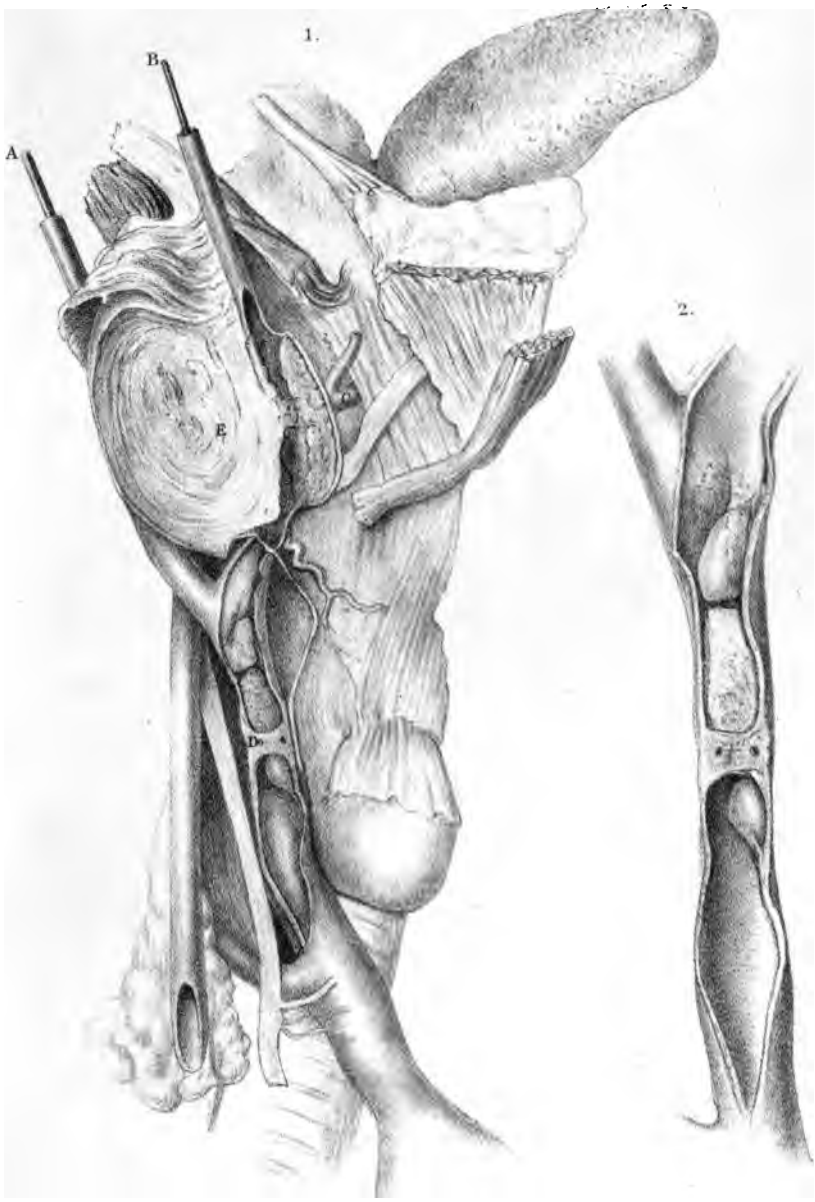
The wall of the aneurism consists of parallel layers of fibrous tissue. In many sections these layers are separated by collections of small round cells; and these sometimes are placed so closely and are arranged with such regularity that they might, under a low power, almost be taken for a distinct coat of the aneurism. The thickest point of the wall seen in the specimen, is produced by a large collection of leucocytes separating the inner and outer layers. The line between the wall and the contents is not sharp. The latter seem for the most part to consist of granular material of reddish colour, with leucocytes scattered sparsely through it. Towards the wall they become more numerous, and in some sections the infiltration of the clot by them is both dense and deep. At the line of meeting of the wall and contents, rather large cells of reddish-yellow colour, from the taking up of red granules, are often seen. A careful drawing of the preparation is appended (see Plate I). In Fig. 2 is given the section of the seat of ligature of twice the natural size, for purposes of comparison with a similar section of a carotid artery tied with a tendon ligature, figured by Mr. C. T. Dent in the sixty-fourth vol. of the 'Medico-Chirurgical Transactions.'

DESCRIPTION OF PLATE I.

Aneurism of the External Carotid Artery, ligature of the Common Carotid, with cure of the Aneurism (CHRISTOPHER HEATH, F.R.C.S.).

FIG. 1. A. Internal carotid.
B. External carotid.
C. Common trunk of lingual and facial artery.
D. Seat of ligature, the divided ends of the silk are seen in the section.
E. Aneurism filled with clot.

FIG. 2. Seat of ligature twice the natural size.



E.S.Gibson del. R.I.Hintern lith.

Mintern Bros. imp.

ANEURISM OF THE ARCH OF THE AORTA;

LIGATURE OF THE RIGHT SUBCLAVIAN AND CAROTID ARTERIES.

DEATH BY RUPTURE OF THE SAC ON THE FIFTY-FIRST DAY AFTER THE OPERATION.

BY

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Received February 11th—Read February 13th, 1883.

F. B—, æt. 30, a shopman, was admitted into St. Bartholomew's Hospital, under my care on January 17th, 1881.

History.—Patient has always believed himself healthy. Has never had syphilis. In March, 1880, he had neuralgia extending from the right side of the top of his head downwards behind the right ear, and along the posterior edge of the sterno-mastoid muscle to the shoulder, and thence on the inner side of the arm to the elbow. A month later (April, 1880) his voice changed to a hoarse whisper. The pain and the aphonia have persisted with varying amount to the present date. Seven weeks before admission he first noticed at the root of his neck a swelling about as large as a bantam's egg. This he says pulsated, and was at first firm, but it gradually became soft and tender on pressure.

On examination a pulsating tumour of the size of a large chestnut was seen, the centre of which was an eighth of an inch above the right sterno-clavicular joint, and about the same distance to the inner side of that landmark. It extended three quarters of an inch upwards, and bulged half way across the supra-sternal notch. Dulness on percussion extended for one inch below the clavicle, but not beyond the middle line. The sternal end of the clavicle and the adjacent part of the manubrium sterni were undergoing absorption, and the clavicle was pushed a little forward. The skin over the swelling was normal. On auscultation Dr. Church could detect no evidence that the tumour extended beyond the limits disclosed by external examination, or any signs of pressure on the trachea or on either bronchus. No bruit could be heard in the swelling. The heart sounds were normal and the heart was not displaced. The pupils were equal. The pulse in the right radial artery was slightly less firm than in the left, and a sphygmographic tracing, taken by Mr. D'Arcy Power, showed the short and sloping up-stroke and the waving down-stroke usually present in aneurism. The right carotid and the superficial temporal pulses beat very feebly compared with those of the left side. He complained of pain in the situations already noticed, and of constant aching extending through the chest from the right nipple to the inferior angle of the scapula. Nothing abnormal could be detected in the venous circulation. The urine was healthy. The situation of the tumour close behind the right sterno-clavicular joint, its apparently limited extent, and the absence of pressure signs within the thorax, suggested an innominate aneurism. Keeping in view the well-known difficulty of distinguishing aneurism of the innominate from aneurism of the arch of the aorta, the tendency of the swelling to extend towards the left convinced those who saw the case with me that the tumour was aortic.

The patient was kept as far as possible at complete rest in the horizontal position, and ordered a spare diet.

In the next few days he complained much of pain in

the tumour, and also in the right side of the neck and shoulder; and the swelling, which was very tender, became more prominent, extending towards the left, until its left border reached nearly to the middle line.

On January 31st, after consultation with Sir James Paget, Mr. Savory, Dr. Church, and others, I placed a ligature on the third part of the right subclavian, and on the right carotid at the level of the cricoid cartilage, using the carbolic spray. The ligature was chromicised catgut kindly sent by Dr. MacEwen of Glasgow. The ligature I passed round the subclavian broke as I tied the knot, but I ought at once to say that Dr. MacEwen, when he furnished the specimen, was not aware it was for so large a vessel as the subclavian, and therefore sent me a medium instead of a full-sized ligature. To make the vessel secure I passed another ligature round it in a line with the first.

Short decalcified bone drainage-tubes were inserted, and the wounds dressed with carbolic gauze. Pulsation in the carotid, when this vessel was exposed, was found to be scarcely stronger than that of a radial artery. No change was observed in the tumour when the vessels were tied.

February 1st.—The pain felt before the operation in the head, neck, and shoulder has almost ceased. But pulsation in the aneurism seems unaltered.

2nd.—Has severe pain in the right side of the neck when he swallows. He looks rather dusky and sweats profusely.

3rd.—Pain still severe in the side of the neck, especially on deglutition. He is still a little dusky. No pulsation in the vessels beyond the ligature.

4th.—On changing the dressings to-day there is found considerable brawny œdema of the right side of the neck, and marked duskiness of the integuments. The aneurism pulsates much less on its anterior aspect than it did before the operation, and the swelling is less prominent in this direction, but it has increased markedly towards the left side, and pulsation now extends considerably beyond the middle line and also higher in the neck in the supra-

sternal notch. Thus the operation appears to have had a beneficial effect on the original dilatation, but it has been followed by rapid extension of the swelling towards the left, and by evidence of obstruction to the return of venous blood towards the heart.

Feb. 7th.—The general condition is satisfactory. But he is still dusky and the brawny state of the side of the neck remains. The aneurism continues to enlarge on the left side, and the whole swelling pulsates more than it did two days ago.

9th.—The subclavian wound is very nearly healed. The carotid incision is suppurating very slightly.

14th.—The general condition is good. Temp. 98·8°, pulse 96. He complains of pain in the right side of the head and neck. He is still dusky. The aneurism is slowly extending higher in the neck and further to the left. The inner third of the right clavicle and a considerable portion of the first rib and of the manubrium have been absorbed. The skin over the aneurism is tense, dark coloured, and thin. No pulsation in the right carotid. Slight pulse in the right radial.

27th.—The aneurism is now very large and prominent. Pulsation is forcible and expansile.

28th.—Last night a slight discharge of dark blood and serum, apparently derived from the aneurism, escaped from the subclavian wound, which, however, had long been closed by a thin scar.

March 22nd.—On the 20th there was a sudden gush of about ten ounces of bright blood through the subclavian incision, and a further hæmorrhage last night. This morning another bleeding occurred from both wounds, to the amount of upwards of a pint, and he died at 5 a.m.

A post-mortem examination was absolutely refused by the patient's relatives, and all that could be made out by enlarging the operation wounds was that the aneurism was of very large size within the thorax, and extended, apparently after partial rupture, nearly as high as the thyroid cartilage.

Remarks.—As no post-mortem could be obtained, the cause of the greatly diminished pulsation of the right carotid cannot be stated with certainty. The presence of this symptom, however, led to some doubt whether the case was suitable for distal ligature; for, as the aneurism was increasing in spite of the occlusion of the carotid, and, as occlusion of the carotid has probably a more powerful influence in checking the growth of aneurism than occlusion of the third part of the subclavian, the gain to be anticipated from distal ligature of the latter vessel was somewhat uncertain. The distal ligature of both vessels, nevertheless, was held to afford the patient a better chance of recovery than any other means of treatment that could be adopted.

Some may be inclined to ask why the carotid was tied, as its pulsation was already hardly to be felt? This step was necessary, because it was uncertain whether the artery was obliterated or merely obstructed by the pressure of an overlying portion of the sac. If the latter condition was present and the artery were not tied, blood might again flow through the vessel, should any diminution in the size of the aneurism take place. Under these circumstances the principle of the distal ligature would have been imperfectly carried out. It will be remembered that in the classical case of Mrs. Denmark who had innominate aneurism, Mr. Wardrop, finding no pulsation in the carotid, tied the third part of the subclavian. The tumour subsided, but pulsation in the carotid speedily returned.

This case incomplete from the absence of post-mortem examination, is offered to the Society as one of a series of examples from which, when sufficiently numerous and varied, a just estimate may be formed of the value of the distal ligature in treating aortic aneurism. For this purpose the present instance is a clear illustration of what other cases have foreshadowed, namely, that the distal ligature may not only sometimes fail to do good, but may also do positive harm by suddenly altering the distribution of the blood pressure; and by throwing it on

some unsound portion of the arch, too weak to bear it, may lead to extension of the aneurismal disease in some new direction. When the patient was admitted, the tumour was placed well to the right of the middle line of the neck, and was most prominent just above the right sterno-clavicular joint, and like many examples of aortic aneurism already on record, bore a close resemblance to aneurism of the innominate. But, although the ligature of the vessels was followed at once by cessation of pain, and during the first few days by marked diminution in both the size and the pulsation of the original swelling, the aneurism was found to grow so rapidly towards the left, that ten days after the operation it had nearly doubled its extent. The patient's face also had become distinctly dusky from obstruction to the return of venous blood, and there was for the first time some dysphagia.

It may be asked whether the ligature clearly accelerated the development of the aneurism, or whether it only failed to check the advance of the disease. There could be no doubt that the former was the case. This was a startling result, and it is necessary to inquire whether it was due, in this instance, to some combination of circumstances so rare as to have no bearing on these cases generally, or whether it was one of the dangers with which we always have to reckon in pursuing this method of treatment. The point is one upon which we have not adequate evidence. Yet the present is not the only case which seems to bear upon it. The carotid and subclavian have now been tied in nine published cases of aneurism of the aorta. These are arranged chronologically in the accompanying table.* One proved fatal in a few hours, and it therefore affords no evidence on the question. Of the remaining seven (my case being set aside), Mr. Maunder's, and Mr. Barwell's first case may be briefly noticed. In Mr. Maunder's case, on the day after the ligature, pulsation in—*i.e.* disturbance of the circulation through—the tumour was so great that venesection was performed for its relief,

* Vide, p. 91.

and on the fourth day also there was "strong pulsation" in the tumour. The patient became cyanosed and unconscious, and died on the fifth day. On inspection, a large clot filled the ascending part of the aorta. It is not improbable that the cyanosis depended on venous obstruction resulting from rapid enlargement of the aneurism, following the ligature. In Mr. Barwell's case the vessels were tied, as post-mortem examination proved, for aneurism of the back part of the ascending aorta, on February 15th, 1879, and on April 2nd Mr. Barwell found the patient moving his left arm freely as he played at draughts. "This discovery," he remarks, "accounted for some pulsation I had observed of late on the left side of the sternum." It is not stated how long the patient had indulged in this imprudence, but it is very material to observe that Mr. Barwell attributed the pulsation to "inherent weakness of the aorta which would not permit it to withstand the current increased in the parts beyond, by tying its two large derivations." On May 12th Mr. Barwell states that the patient was suffering from pressure symptoms, and that the aorta was evidently becoming dilated. At the patient's death, sixteen months after the operation, a large aneurism was found involving the transverse arch—the aneurism for which the vessels were tied being, as already mentioned, at the back of the ascending portion. The precise bearing of Mr. Barwell's case on the point under discussion, is rendered doubtful by the fact that the ligature placed on the carotid was tied so loosely that the vessel was not obliterated, and this circumstance must have tended to diminish the strain to which the arch beyond the origin of the innominata would have otherwise been subject. It is clear, notwithstanding, that within some five weeks of the operation, so large a dilatation of the arch had occurred that pulsation could be felt to the left of the sternum. When it is remembered that in cases of aneurism the whole aorta is frequently not only atheromatous, but already considerably dilated, while a second, or even a third aneurism may be present, the

existence of which it is impossible to ascertain, the effect of distal ligature would appear to be a matter of much uncertainty.

This, however, is by no means a conclusive argument against this method of treatment, for, on the other side, is the well-established fact, that in some of the cases the patients have received very marked relief, and have had their lives considerably prolonged. Dr. Lediard's patient, who was very nearly asphyxiated before the operation, lived for nine months, and for four months was actively about, and enjoying excellent health.

Mr. Barwell's patient lived for sixteen months, and the pressure signs within the thorax almost disappeared; while Mr. Heath's case survived for four years, and for two years was free from any increase of the disease, although she was a confirmed drunkard, and utterly reckless of her health. Thus, though no case has, so far as we know, yet been cured, some have been distinctly relieved, and have some prolongation of life secured to them by the operation.

The practice of tying the carotid and subclavian for aneurism of the aorta originated, one may almost say, fortuitously; for in the first three cases in which it was undertaken the operators believed they were dealing not with aortic but with innominate disease. Nevertheless it was from two of these cases—Heath's and Sand's—but especially from Heath's, that we learnt the fact that this operation may be followed by very favourable changes in the condition of the patient. It was after this preliminary demonstration that Mr. Barwell, in 1879, tied the vessels for what he was aware was an aneurism of the aorta. Since this case occurred those of Dr. Lediard, and Dr. Wyeth have been recorded, and in all the operation was, beyond question, followed by markedly beneficial results. Surgeons have thus been led on from case to case; but this course has been taken tentatively; and spite of pre-existing notions. Besides the accidental results of clinical experience there has been very little to guide the surgeon,

and no generally recognised principle to rest upon. We have certainly been carried far beyond the suggestion of Brasdor, and far beyond the practice of Wardrop. It thus behoves us to tread cautiously on this new ground. Of the cases in the table, Green's must be passed over, for full particulars have not been published. Barwell's second case proved fatal in thirty hours, and must also be set aside. Wyeth's patient was alive on December 25th, 1880, and no subsequent report has been made.

Let us glance at the remainder. Heath's, Sand's, and Lediard's, were all examples of simultaneous occlusion of the carotid and subclavian; the patients were distinctly better after the operation, and they survived for a considerable period. In both Heath's and Lediard's the carotid was closed, but the first and second parts of the subclavian remained fully patent. These cases suggest, so far as they go, that it would be best to tie the carotid, and refrain from ligature of the subclavian; seeing that, as its first and second divisions remain patent, the ligature of its third part would, *primâ facie*, seem to have little effect. But, in the case just related, the carotid was very nearly closed before the ligature was applied; and therefore whatever influence the occlusion of this vessel was calculated to exert was already in action; consequently the case was one of ligature of the subclavian only. Yet, so far from this operation having no effect, the immediate result was rapid enlargement of the aneurism. In Mr. Barwell's first case (the preparation is in the museum of the College of Surgeons) the carotid was tied so loosely that it remained fully patent. While, as in the other cases, the first and second parts of the subclavian continued widely open. In other words, both these vessels continued to transmit very nearly, if not quite, their full arterial stream. We have thus three conditions under which the distal ligature has seemed to have a marked effect. 1. When the carotid, and the third part of the subclavian have been simultaneously occluded, the first and second parts of the subclavian, however, remaining patent. 2. When the third

part of the subclavian has been tied, the carotid being already practically closed. 3. When the third part of subclavian has been tied, the carotid though tied, remaining freely open. It may also be mentioned that, in the well known case, recorded in his very able pamphlet, by Dr. Cockle, Mr. Heath tied the left carotid, with very marked benefit to the patient, for what, as the post-mortem four and a half years later showed, was aneurism of the ascending part of the aorta. It is difficult to see how the effect observed in these various cases was produced. Many surgeons, bearing in mind the well-known fact that patients with aortic aneurism may survive for a considerable period, and may improve in a very unexpected manner even when their condition has become desperate, if they can be persuaded to maintain absolute rest, attribute the relief which has followed distal ligature, more to the quietude which the operation has been the means of enforcing, than to the direct effect of the operation itself. This view is not consonant with the facts of some of the cases. In some of the cases complete rest had been enforced and had failed; whereas the effect of the operation was immediate and unmistakable. Although it acted prejudicially in the case under consideration, the effect of the operation—though this was practically merely ligature of the subclavian—was perfectly distinct. While in Mr. Barwell's, though the carotid was not closed, the ligature of the subclavian was followed not only by improvement in the original aneurism but also, very quickly, by symptoms of dilatation of the arch towards the left. Again, in Mr. Maunder's case the operation was rapidly followed by cyanosis, and other signs of increased venous obstruction, as well as by strong pulsation in the sac (in this instance the aneurism was found to be seated on the arch beyond the origin of the innominate). The true explanation, perhaps, lies in another direction. All surgeons are aware by what slight mechanical interference aneurisms are sometimes cured, whether by pressure on the proximal, or even on the distal side, or by means of

flexion, or Esmarch's bandage. Possibly such temporary improvement as has been obtained in the cases under discussion may have been due to the fact, not hitherto fully recognised, and certainly not anticipated, that interference of even a limited character may have a powerful effect—though not always for good—on this form of aneurism. But without theorising upon the subject, the opinion may be expressed that the value of distal ligature for aneurisms of the aorta must be regarded as still *sub judice*, and that there are some cases in which it may be attended with very grave results.

No.	Operator and reference.	Date.	Age.	Sex.	Period of survival.	Cause of death.
1	Heath, 'Lancet,' Jan. 5, 1867, July 2, 1870	1865	30	F.	4 years	Bursting of sac.
2	Maunder, 'Surgery of the Arteries'	1867	37	M.	5 days	Occlusion of ascending aorta by clot? Patient became cyanosed and unconscious.
3	Sands, 'Bellevue Hosp. Reports,' 1868-69	1868	43	F.	13 months	Pressure of tumour.
4	Green, Wyeth's Pamphlet, New York, 1880	1874	45	M.	3 months	Bursting of sac.
5	Barwell, 'Med.-Chir. Trans.,' vol. lxii, p. 393	1879	36	M.	16½ months	Wearing out. (*Vide* Wyeth's Pamphlet.)
6	Lediard, 'Med.-Chir. Trans.,' vol. lxiv, p. 226	1880 March	42	M.	9 months	Bursting of sac.
7	Wyeth, Pamphlet	1880 Sept.	42	F.	Living Dec., 1880	—
8	Barwell, 'Med.-Chir. Trans.,' vol. lxiv, p. 226	1880 Oct.	37	M.	30 hours	Pneumonia.
9	Marsh	1881	30	M.	51 days	Bursting of sac.

A CASE

OF

ANEURISM OF THE ARCH OF THE AORTA

INVOLVING THE

INNOMINATE ARTERY:

WITH REMARKS ON THE DISTAL LIGATURE.

BY

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Received February 6th—Read February 13th, 1883.

THE following case presents features which seemed to encourage the operation of ligature of the common carotid. It also shows the untrustworthiness of any conclusions, either as to the operation to adopt, or as to the anatomical relations of the aneurism, drawn from the fact that distal pressure on one of the large arteries in connection with the tumour diminishes the force of the aneurismal pulsation.

Sarah B—, a married woman, æt. 43, a "farm labourer," was admitted into the Middlesex Hospital on September 7th, 1882.

History.—Six months ago she noticed a severe gnawing pain across the front of the sternum which she attributed to indigestion. Three months ago she began to feel after exertion "a great throbbing and beating" just above the inner end of the right collar bone, which became worse by degrees. Quite lately she had felt pain shooting up the right side of the neck to the ear, and through the thick part of the shoulder. She had no cough, no dysphagia, had never had syphilis, and her general health had always been good.

State on admission.—A dark, fresh-complexioned, spare woman, with an expansile pulsating tumour plainly seen as well as felt above the right sterno-clavicular joint and inner fifth of the right clavicle. The upper limit of pulsation was fully an inch and a quarter above the upper border of the clavicle; the inner limit reached the median line of the neck, the outer corresponded to the outer border of the sterno-mastoid. Pulsation was also felt in the first and second intercostal spaces for at least an inch outwards from the right edge of the sternum. The inner half of the clavicle rose and fell with the aneurism, and its sternal extremity looked partially dislocated forwards. There was a loud bruit over the whole of the tumour. The external jugular and other veins on the right side of the neck and upper part of the chest were dilated. There was no irregularity of the pupils, no appreciable difference in the pulse at the wrists (or, if any, it was the left that was slightly weaker), nor in the facials, but the right temporal was more feeble than the left. By compressing the trunk of the right common carotid a distinct diminution in the force and fulness of the pulsation in the aneurism was produced—this was quite marked and readily appreciated by the eye and finger. Compression on the third part of the subclavian caused no difference in the aneurism. The tissues over the cervical part of the tumour were very thin. The pulse was small and weak, 116 when sitting and 104 when lying down.

I resolved on trying Tufnell's treatment, and ordered one

grain of acetate of lead in a pill three times a day. On this treatment she improved, her pulse dropped, so that on the 9th of September it was 88, on the 11th 80, on the 13th 70, and from this time it continued to be between 70 and 76; it was also regular, compressible, and small. The aneurism also improved. By September 19th the clavicle was no longer raised by the pulsation, and the appearance of luxation at the sterno-clavicular joint had gone.

So she continued till October 4th, when she complained of giddiness and pain in the head; the aneurism pulsated more than it had done for many days previously, and the inner end of the clavicle again rose and fell with the pulsation. Between this date and October 20th the tumour became softer and extended further to the left, reaching beneath the border of the left sterno-mastoid. Its impulse was more plainly felt in the first intercostal space than it had been, and there was some slight bulging of the upper space and first two cartilages of the right side.

On October 20th, after consultation with Dr. Powell, iodide of potassium in twenty-grain doses three times a day was ordered, and the pills were of course discontinued. The diet was to remain the same. Up to this time there had been no symptom whatever of plumbism, though the blue line on the gums had been looked for several times; but, after taking the iodide of potassium for nine days she developed the blue line, experienced colicky pains in the abdomen, loss of appetite and sickness. The iodide was therefore discontinued on 31st October, and the restricted diet on November 2nd.

By November 14th all symptoms of plumbism had disappeared except the blue line, and this was fast fading. The condition of the aneurism was about the same. She was allowed to sit up to-day for the first time since treatment was commenced. It was now noticed that the pulsation along the carotid artery was feebler, but distal compression still produced an appreciable effect on the aneurism.

On November 25th, at 2.30 p.m., ether was administered

and the operation of ligaturing the carotid was commenced. After the usual incision for applying the ligature on the level of the omohyoid, what looked like the sheath of the cervical vessels was opened and a feebly pulsating vessel smaller and thinner than a normal common carotid, but with none of the appearance of a large vein was brought into view. No other vessel could be found. The pneumogastric nerve lay to the inner side of the vessel upon a quantity of sclerosed fibrous tissue. Whilst the vessel was being held aside for the purpose of examining this tissue, it was injured and a rush of venous blood flooded the wound, flowing as from a vein. Torsion forceps secured the opening and controlled the hæmorrhage, and a further examination of the parts was made without discovering the artery. I then dissected the wounded vein downwards towards the aneurism, and found it pulsating *forcibly* near the sac. I tied the vein above and below the wound in it with ox aorta ligature, very kindly supplied to me by Mr. Barwell. On tightening the lower ligature the aneurismal pulsation diminished somewhat and the knot of the ligature moved forcibly with each beat of the aneurism.

For several hours after the operation the patient lay in a semi-conscious state, but nothing requiring notice occurred after she rallied until the morning of the 27th, when the right side of the face was looking waxy and œdematous, and she complained of pain over the lower part of the right side of the neck. There was considerable œdema and redness between the clavicle and trapezius, but the wound looked united and the tissues immediately around it were quite natural in appearance. During the next two days the œdema and pain increased. Subsequently matter formed and burrowed inwards to the wound and downwards to the upper edge of the great pectoral muscle; incisions were made, and the wound opened up, and a quantity of pus was discharged. There were no rigors, and very slight constitutional disturbance until two days before death, when the respirations and pulse were rapid, the

temperature rose to 103·6°, suppuration became very free, the aneurismal pulsation increased, and the patient rapidly sank and died of asthenia on December 9th.

A post-mortem examination was made on December 11th by Dr. Fowler, and the aneurism, aorta, and large vessels of the neck were subsequently carefully dissected by Mr. Sutton.

The following notes are extracted from the post-mortem report:

There were the evidences of diffused suppuration between the skin and platysma, and beneath the platysma, in the right posterior triangle of the neck. The suppurating areas communicated with the exterior through three incisions, as well as through the operation wound. The aorta, from the semilunar valves to about half an inch beyond the origin of the left subclavian, was dilated, and measured (after being in spirit several days) 3§ inches in circumference at the junction of the ascending and transverse portions of the arch. The tunica intima was much thickened, and dotted with patches of atheroma.

At the point of origin of the innominate artery and extending a little to its right side was a circular opening in the aorta 1¾ inches in circumference, and therefore larger than the normal orifice of the innominate. This opening led into a sacculated aneurism of the arch of the aorta, as well as into the innominate vessel, the lower end of the right wall of which was deficient for half an inch and presented a crescentic, rounded, thickened edge.

The sac of the aneurism was oval, and measured (after contraction in spirit) about 1¾ inches vertically, by 1 across; it contained a small quantity of laminated clot about its base, elsewhere there was little or none. The postero-internal wall of the sac was formed partly by the innominate artery, the external coat of which had been raised upwards by the growth of the aneurism for a distance of ⅛ of an inch; thus, on looking into the aneurism, a concave triangular shaped area was seen at this point where the wall of the artery was exposed.

The innominate vessel from origin to bifurcation, as well as the commencements of the right subclavian and common carotid arteries, were behind the aneurism. Both subclavian and common carotid were considerably dilated at their origin (the subclavian for the first inch of its course), and their coats, as well as those of the innominate, were much thickened.

The common carotid was firmly bound down by a dense, thick, layer of sclerosed connective tissue, which stretched from the sac of the aneurism as high as the upper border of the larynx. The vessel was thus completely flattened and shut out of view, and it was only after several minutes of difficult dissection, with the parts all laid bare, that it could be found. At a distance of 1⅝ inch from its origin the artery was completely occluded, all attempts to pass the finest probe through it being ineffective. This occlusion continued for ⅛ inch, when the vessel became again pervious. Just above the occlusion the carotid was somewhat dilated, as it was also near its bifurcation.

The right internal jugular vein bore two ligatures about an inch apart, opposite the larynx; the lower ligature moved up and down upon the collapsed vein like a ring; the upper one became detached during dissection. The lower part of the vein was intimately incorporated with the right wall of the aneurism, but there was a free channel through it into the right innominate vein. Decolorised clot extended from the lower ligature into the portion of the jugular adherent to the sac. No communication existed between vein and aneurism. The left innominate vein was completely occluded; so that after the right internal jugular was tied, all blood from the head and neck must have returned by the right external jugular and small anastomotic branches. The pneumogastric nerve was closely adherent to the back of the aneurism on its right side.

The other viscera were examined, but require no notice.

Remarks.—The case illustrates the extreme difficulty of diagnosing some forms of aortic aneurism, from aneurism

of the innominate. No one who examined this woman questioned that the aneurism was innominate, and some very capable diagnosticians considered it to be a simple sacculated aneurism of that vessel. Even after dissection it was impossible to make out its true character and connection until the sac had been laid freely open in front, and the innominate artery behind.

The situation and outline of the tumour; the pain in the shoulder and over the right side of the head and neck; the *shooting* character of the pain, and the venous congestion of the right side of the neck led to the diagnosis of innominate aneurism. The origin of the disease from the aorta might have been suspected if more weight had been given to the severe *gnawing* pain across the front of the *chest* suffered at the outset; to the dilated veins on the right side of the upper part of the chest; to the equality of the radial pulses; to the absence of any cough, dyspnœa, and throat dryness, of any deflection of the trachea, of any numbness or loss of power in the right arm,—such frequent symptoms in innominate aneurism; and to the fact that aortic aneurisms cause tumour in the neck. When, however, characteristic symptoms are blended as they were in this case, a precise diagnosis is not possible.

The marked effect on the aneurism of pressure on the common carotid, and the negative effect of compressing the third part of the subclavian are features of great importance. Possibly it was in part due to the closer and greater length of contact of the carotid with the sac; but after events lead me to think that the chief effect is probably attributable to the simultaneous compression of the pneumogastric trunk. Sir William Fergusson supposed that the pressure caused its effect by its influence on the brain and on the action of the heart, because similar symptoms were produced in his case of innominate aneurism by pressing on the left carotid.[1]

[1] Sir Wm. Fergusson, 'Lond. and Edinb. Monthly Journ.,' Nov., 1841, p. 793.

The great diminution in the pulsation of the aneurism by distal pressure on the common carotid decided me to tie that vessel alone, with the hope that laminated clot would extend from its orifice into the sac. The case, however, like others, shows how little value can be attached to a difference in the effect of pressure on the carotid, and on the subclavian.[1] Sir William Fergusson was led to tie the carotid alone, yet his case turned out to be an aneurism of the innominate involving the subclavian. In Mr. Holmes's[2] case of aortic aneurism involving the innominate, pressure on the subclavian produced the greater effect, and on this account, as he mistrusts the ligature of the third part of the subclavian alone, he simultaneously ligatured it and the carotid; yet after death "the arteries opening out of the tumour seemed healthy," and there was no extension of clot into the aneurism from either of them. The anatomical relation of the subclavian artery to the aneurism in my case does not lead me to suppose that the aneurism would have been cured, or even in any degree improved, if the subclavian, even in the first part of its course as well as the carotid, had been successfully ligatured.

Nature sometimes performs for us an experiment, equivalent to the double ligature, without effecting thereby the cure of the aneurism. Thus Dr. Douglas Powell has

[1] With reference to the effects of pressure on the carotid artery, my attention has been drawn by Mr. Roger Williams to the following interesting remarks by Dr. M. Moir in his 'Outlines of the Ancient History of Medicine' (1831, p. 138). Speaking of Rufus, the Ephesian, one of Galen's predecessors, who died in the reign of Trajan, Dr. Moir says, "In his anatomical treatise he mentions the recurrent nerve, which had then been newly discovered; and, in the same, makes the remark that pressure on the carotid arteries had been observed to take away the voice. He points out, however, the true cause of this fact, by showing that it does not arise from pressure on the arteries, but on the nerves, which are contiguous to them." Portal ('Histoire de l'Anatomie,' tome i, p. 74) has also quoted this same observation of Rufus.

See also Mr. Savory on the influence of pressure on the pneumogastric nerve in the neck ('Lancet,' June 9, 1883).

[2] 'Lancet,' 1872. Holmes Lectures on Aneurism.

recorded a case in the twentieth volume of the Pathological Society's 'Transactions,' p. 118, of an aneurism of the arch of the aorta implicating the whole length of the innominate, in which, after death, it was ascertained that both the carotid and origin of the subclavian were quite obliterated, and yet scarcely any laminated clot existed in the aneurism. Dr. Powell's case was peculiarly like mine; and in his, as in mine, it appeared at first sight, on dissection, that the aneurism was connected with "an aorta from which the subclavian and right common carotid were given off as separate trunks."

It does not seem from the cases tabulated by authors and those more recently made public, that the distal ligature affords much encouragement to the surgeon in *innominate* aneurism. I do not gather from the records of these cases that there is a single instance of *complete cure* after ligature of the subclavian alone; or ligature of both vessels simultaneously or consecutively; and only one case, namely, Evan's, after ligature of the carotid alone. The great majority of the operations have been complete failures; though a few cases have been temporarily benefited. Bryant's, Wardrop's, and Broca's cases, in which the subclavian only was tied, improved, and the patients lived three years, two years and a quarter, and seven months respectively. Of the cases in which the carotid alone was tied, Morrison's and Mott's were relieved, and the patients lived twenty months and seven months respectively, whilst one of Pirogoff's cases recovered from the operation but was not cured of his disease. Again, of the cases in which both vessels were ligatured, all died within periods varying from several hours to four months (Fearn's), with the exception of Langley Browne's,[1] which was probably of traumatic origin, and Barwell's three cases all of which were much relieved by the operation, though all have since died.

[1] 'Med.-Chir. Trans.,' vol. lxv, p. 21. Browne's case lived about one year. No post-mortem.

In estimating the value of the distal ligature, too much importance has perhaps been attached to cases such as Fearn's, Morrison's, and others, in which, after death, the aneurism has been found filled with clot.[1] As distinct from the effects of ligature, and with regard to the formation of clot, some importance, ought to be given to the condition of the blood and general state of circulation brought about by the fatal disease, as well as to the slow approach of death; circumstances which in themselves favour, in no small degree, the deposition of laminated fibrine.

As regards *aortic* aneurisms, I think it may be questioned whether Mr. Heath's well-known case of double distal ligature, which hitherto has proved quite exceptional in its success, has not placed Wardrop's operation in a light altogether more favourable than subsequent experience warrants. This opinion, I venture to think, is not invalidated by such cases of double distal ligature for aortic disease as Barwell's (two), and Lediard's, whose patients died at the end of seventeen, fifteen, and nine months after the operation respectively; or of Wyeth's, whose patient recovered from the operation and was alive three months after it, but with the aneurism still uncured.

Such cases as Rigen's, Tillanus's, Holmes's, and Heath's, of ligature of the *left* common carotid for aortic aneurism ought not, I would submit, to be weighed in the same balance as the operation on the right side. The operation on the left vessel, in suitable cases, gives the patient a much better chance of cure, because the ligature has nearly all the advantages, without the risk, of ligature of the innominate for aneurism of the right part of the arch. It is Brasdor's operation; whilst there is only half the risk and a third more advantage than in the double distal ligature.

[1] That laminated clot is not tantamount to cure, but is only a step in the process of cure, of an aneurism is not sufficiently appreciated: but this is proved by many cases on record, and is to be seen from time to time in the post-mortem room, in instances in which, though the aneurism is almost if not quite full of laminated clot, the patients, nevertheless, have died from hæmorrhage owing to the giving way of the sac.

It will, perhaps, be admitted that when the tumour is not limited to the innominate and its derivatives, but is partly or entirely aortic, the chances of complete obliteration of the sac by the double ligature are much diminished. If the enormous difficulty of diagnosis, the great risks of the operation, the possibility of spontaneous improvement, if not of cure, and of palliation by rest and diet and also the fatal results of recorded operations, be taken into due consideration, it seems that the distal ligature on the right side should be limited to desperate cases, and then performed only with the expectation of relief, not of cure: and that as ligature of the carotid has appeared to take a larger share in the consolidation of the aneurism, its effects should be tried before the patient is submitted to the further risk of the operation on the subclavian.

The pulsation in the internal jugular vein due to its connection with the sac of the aneurism is a point of some clinical importance. Though the pulsation along the course of the carotid was for some time before the operation observed to be feebler than at first, yet, as a pulse was perceived, as it was supposed, in the vessel, and as compression along the course of the carotid diminished the aneurismal pulsation and further as constitutional treatment had not succeeded, ligation of the carotid seemed to be indicated. Occlusion of the artery had been accomplished, as it proved, long before the operation was undertaken, but no good came of this spontaneous occlusion; the aneurism, on the contrary, continued to enlarge after the carotid was plugged. Probably the date at which the artery was blocked was between the 4th and 20th of October; certainly the plug had existed a long time before death.

The development of lead-poisoning by the administration of iodide of potassium is noteworthy, and tends to confirm the views expressed by Dr. Fagge in a paper in the fifty-seventh volume of the 'Med.-Chir. Transactions,' except, however, in this particular: that the blue line had not existed during the time the lead pills were taken,

and appeared only after the iodide of potassium had been commenced.

The condition of the ligatures at the time of death was to all appearances the same as at the time of their application. The specimens of ox-aorta used were found to be brittle as well as elastic, but still they were capable of being tied tightly enough to accomplish their purpose.

I am indebted to Mr. Roger Williams for the following account of the states of the ligatures after the patient's death:

"The noose of one of the ligatures was found to be loose on the included vessel, which had shrunk within it. There were no adhesions between it and the adjacent structures. The knot held as firmly as it had been tied at the operation. To unfasten it was easy, as the several parts in contact were not firmly adherent. After removal, the ligature had a soapy feel; but otherwise it presented no obvious alteration.

"Microscopical examination showed the whole thickness to be infiltrated with granular leucocytes. These were for the most part arranged in bead-like chains between the fibre bundles of the tissue; the latter were evidently in a state of commencing disintegration, presenting where in contact with the chains of leucocytes minute crenated erosions; moreover, these bundles also showed signs of granular degeneration, and in many parts of the specimens they were splintered into short fragments, and in a few they were quite destroyed. A small number of giant cells were detected in the tissue, in one of them I counted thirteen nuclei."

To summarise: the case shows (1) the impossibility of diagnosing with accuracy some forms of aortic from innominate aneurism; (2) the untrustworthiness of any conclusion drawn from the effects upon the aneurism of distal compression of the large arteries; (3) that a very appreciable amount of pulsation may be felt in the jugular vein when it has become adherent to the sac of the aneurism,

and that this may lead to error if the carotid has become occluded; (4) that the anatomical position of some aortic aneurisms is such that they are not likely to be cured by the occlusion of both subclavian and carotid arteries; and (5) that, as Dr. Fagge has pointed out, iodide of potassium is capable of developing plumbism in persons in whose system the lead is lying inactive.

DESCRIPTION OF PLATES II AND III.

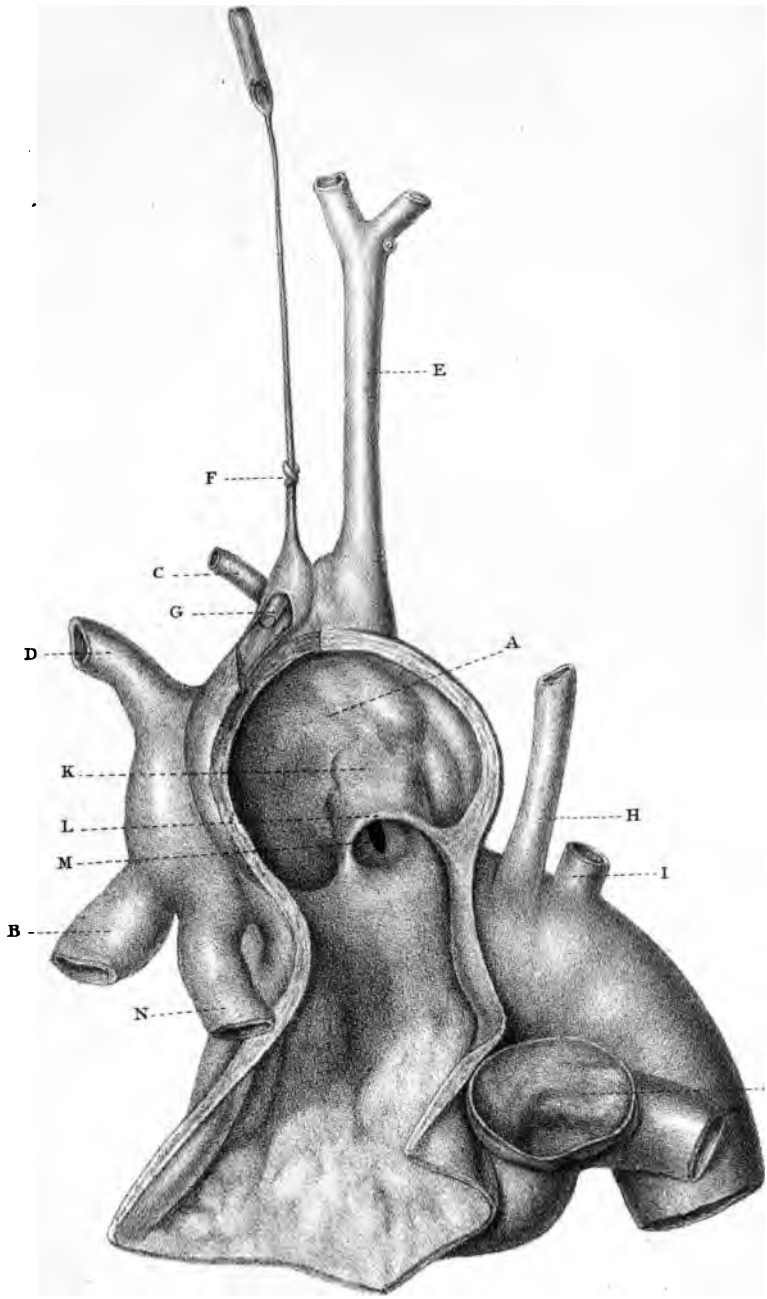
Aneurism of the arch of the aorta involving the innominate artery (HENRY MORRIS, M.A., F.R.C.S.).

PLATE II.—Aneurism of the arch of the aorta involving the innominate artery. Anterior view.

A. Sac of aneurism stretched open.
B. Superior vena cava.
C. Right subclavian artery.
D. Right subclavian vein.
E. Right common carotid artery.
F. Internal jugular vein, knot of ligature *in situ*.
G. Clot in internal jugular below point of ligature, and in the part of the vein adherent to the sac of the aneurism.
H. Left common carotid.
I. Left subclavian artery.
J. Pulmonary artery.
K. Right wall of innominate artery forming part of wall of aneurism.
L. Semilunar margin of right wall of innominate.
M. Small aneurismal pouch on posterior and left side of innominate.
N. Left innominate vein thrown over to right side.

PLATE III.—Ditto. Posterior view.

A. Innominate artery laid open behind and exposing the two pouch-like dilatations in its wall, and the rods passing into the carotid and subclavian arteries.
B. Superior vena cava.
C. Subclavian artery.
D. Right vertebral artery.
E. Dilated origin of right common carotid.
F. Knot of ligature on internal jugular vein.
G. Internal jugular vein laid open just below where it leaves the wall of the aneurism, and as it joins right subclavian vein.
H. Left carotid.
I. Left subclavian.
J. Right pulmonary artery.
K. Opening from innominate into sac of aneurism.



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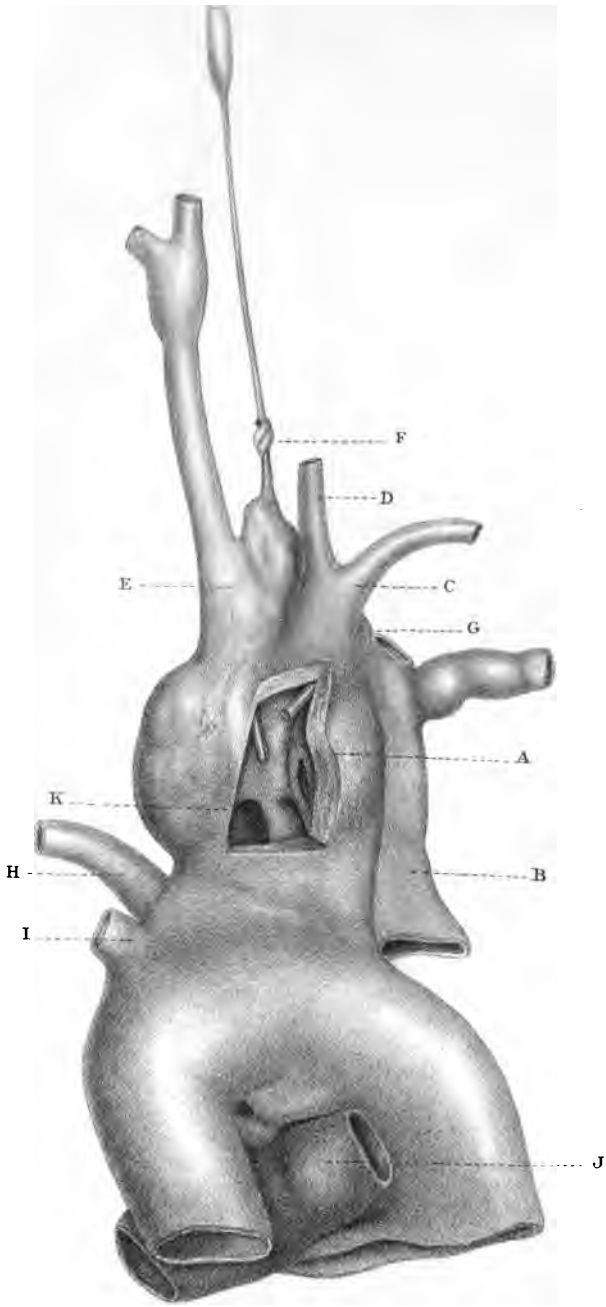

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OPHTHALMOPLEGIA EXTERNA

COMPLICATING A CASE OF GRAVES' DISEASE.

BY

FRANCIS WARNER, M.D.,

ASSISTANT PHYSICIAN TO THE LONDON HOSPITAL AND TO THE EAST LONDON HOSPITAL FOR CHILDREN.

Received May 9th—Read October 24th, 1882.

MARION H—, æt. 25, was admitted into the London Hospital, March 22nd, 1881, with the signs of Graves' disease well marked. She enjoyed good health till February, 1877, when the catamenia became scanty. In November of that year she was admitted in-patient under Sir Andrew Clark for tonsillitis, and was then found to present exophthalmos with considerable enlargement of the thyroid, especially in the right lobe. In 1878 she was again an in-patient for Graves' disease, and was troubled with palpitation, dyspnœa, bronchitis, and slight blood spitting; the temperature sometimes rose as high as 103° F. without any inflammatory cause. During 1878-9 there were many attacks of dyspnœa, and she was often distressed by thirst and night sweating; during this period she grew stout but pale.

She continued as a hospital nurse, but suffered much from headache, vertigo, and palpitation, as well as from intercurrent attacks of diarrhœa; also the legs were often swollen. Menstruation was absent from November, 1880, to October, 1881, when it became re-established. She first suffered from temporary diplopia about January,

1880; this lasted only a few weeks, and entirely passed away as the general health improved. In November, 1880, she first began to notice that she had to turn her head to look at an object, being unable to move the eyes properly. When admitted in-patient in March, 1881, she was excessively nervous, very excitable and irritable in temper; she was, therefore, at her own request kept in bed, almost in darkness, with screens around her. She complained of double vision and "flies before her eyes," which appeared to her so real that she attempted to catch them; she was also troubled with giddiness, thirst, constant picking of the lips, and palpitation, so strong that the whole body shook with the throes of the heart, accompanied with dyspnœa. The signs of Graves' disease were well marked, but not excessive; the usual complications—diarrhœa, tonsillitis, bronchitis, amenorrhœa, and headache—were very troublesome. The right lobe of the goitre was much larger than the left, and a souffle was heard over it; the circumference of the neck over the goitre was fourteen inches; there was no cardiac bruit.

To epitomise the symptoms which occurred during the seven months the patient was under observation: there were frequent attacks characterised by palpitation, dyspnœa, head pain, insomnia, and vomiting, frequently attended with pyrexia rising to 102° F., also gastric crises, marked by vomiting, diarrhœa, epigastric tenderness, blood spitting, and thirst. During April she often complained of throbbing pain in the temples and palpitation with the gastric attacks, but these recurred less frequently and with less severity, so that towards the end of the month there was marked improvement, less dyspnœa, she was quieter, there was less irritability of temper, and more self control over language, &c., but she still required screens around her bed. The muscles of expression were found to be weakened on both sides, so that the face was very expressionless. When the patient laughed some irregularity in the movements of the mouth was detected, but there was no definite facial paralysis.

Early in April she was troubled with corneitis, attended with great photophobia, which attacked the left eye principally, and lasted nine days; a relapse of corneitis occurred in the right eye for five days, attended with a stye in the lower lid, each attack of corneitis being attended with "a gastric crisis."

Mr. James Adams kindly saw the patient with me, and we took the following note:—Diplopia, external strabismus (very slight), proptosis, traces of corneal ulceration. The movements of either eye were very deficient; in the right eye there was vertical movement through about 4°, but in the horizontal plane not more than 2°; it was also specially noted that the left eye, while it could move outwards, had no power of inward movement. The pupils were both sluggish in movement, but reacted to light and accommodation, the left acting better than the right. Either eye could read type J. 1 at six inches.

During May there were several gastric crises with severe headache. Examination for spinal symptoms showed the absence of the signs of ataxy, and the knee jerk was normal. As she lay in bed there was distinct tremor of the feet, increased by any mental excitement, and this remained constant. The legs were slightly œdematous. Pulsation in the thyroid diminished, and the general condition improved, but the goitre did not lessen in size. The condition of the eyes continued to distress the patient; there was great photophobia caused by ulceration of both corneæ, which occurred principally on their lower segments, and was probably due to exposure at night following upon the weakened condition of the facial muscles; there was proptosis, together with palsy of the fifth pair of nerves; the left eye again being the worst in extent of ulceration and amount of pain. Iced compresses were applied during the day, and a bandage at night; a leech placed behind the ear gave relief to the pain and conjunctivitis on that side.

During June the general comfort of the patient con-

siderably improved, the gastric crises stopped, but palpitation continued troublesome, the thyroid did not now visibly pulsate, and the carotids were not seen throbbing; the circumference of the neck over the goitre measured fourteen inches as before. Tactile and thermal sensibility were much lessened in the face, and they were blunted throughout the whole body; smell and taste were unimpaired. Œdema of the legs continued; an ulcer formed over the left internal malleolus, but healed in four days. Early in the month Mr. Couper examined the patient, and reported both fundi oculi normal, refraction myopic, no spontaneous pulsation in the retinal vessels; partial double ptosis, and some divergent strabismus with diplopia. There was some feeble movement of the eyes, but the condition approached almost complete double ophthalmoplegia externa, though in neither eye was the movement in any direction completely lost; conjunctivitis continued, but was less intense; photophobia was troublesome. The double ptosis increased till the palpebral fissure was not more than three lines; the eyeballs were anæsthetic, so that the patient could only just perceive a touch upon either cornea. There was no convergence for near vision.

In July the gastric crises returned with delirium at night; there was some general muscular weakness, and the paresis was so marked in the masticatory muscles that the patient could not bite one's finger so as to hurt it; the tremor of the feet continued. At the middle of the month Litres' cold irrigator was applied to the head and the goitre, with very good results; in a fortnight the proptosis had diminished, and the goitre was lessened, so that the circumference of the neck had fallen from fourteen to thirteen inches. As to the eyes there was distinctly some power of movement upwards and downwards, but none in the horizontal direction. From the end of July till the time of her discharge in November the general health greatly improved, she gained in weight and in strength, the diarrhœa ceased, the headaches,

gastric crises, and rises of temperature became infrequent and very transitory.

In August she was ordered as follows:

℞ Liq. Hydrarg. Perchlor., ʒss;
Potass. Iodid., gr. v;
Tr. Card. co., ʒj;
Aquæ ad ℥j, ter d. s.

Slight mercurialism followed. The iodide was increased to gr. xv, and this was continued till the end of August, when she was ordered a simple tonic. There appeared to be no evidence of the presence of syphilis. Menstruation returned in September, and has continued regular since; it had been absent for eleven months. The goitre entirely disappeared, the circumference of the neck falling from fourteen to twelve inches and a half.

In January, 1882, Mr. Tweedy kindly examined the patient and reported:—"Pupils fairly active and in a state of medium dilatation, both eyeballs prominent, especially the left, the right the better sighted. On the left cornea at its lowest part is a linear horizontal scar corresponding to the palpebral fissure; the pupillary edge of the iris is jagged at the inner part, especially above. Just opposite the upper and inner edge is a clot of uvea on the anterior capsule of the lens, the remains of old iritis. Optic discs and fundi normal. Very little movement of the globes, rather more in the vertical direction than in the horizontal; the left eye moves a little more than the right."

In this case the immobility of the eyes was never absolute in any direction, and the limitation of the range of movements was not equal in all directions. Reasons for thinking the paralysis of the ocular muscles central and not local in certain nerves are as follows: the average symmetry of the lesion, paralysis of the third, fourth, and sixth pairs of nerves would not account for the greater limitation of the movements in horizontal as compared with the vertical planes.

The symptoms and pathological signs in this case may be classified as those dependent upon the Graves' disease, its complications, and the ophthalmoplegia externa; in the former group may be placed the exophthalmos, goitre, palpitation with gastric crises, headaches, and dyspnœa, then the general mental excitement, vertigo, tremor of the lower extremities, weakness of the fifth and seventh pairs of nerves, with general reduction of sensibility.

In most points this case corresponds with Mr. Hutchinson's description of ophthalmoplegia externa given in vol. lxii of the 'Transactions.' Agreement is seen in the double ptosis, a want of exact correspondence in the conditions of mobility of the two eyes, the paresis of the fifth pair of nerves, and the good vision. In six of Mr. Hutchinson's cases the lower extremities were weak, with a condition more or less approaching ataxy. In this case there was tremor of the feet without defect in walking. Two of Mr. Hutchinson's cases presented the one, insanity, the other, violent mental excitement; in the case narrated the mental excitability was marked. A difference is found in bilateral weakness of the muscles of the face.

It is noteworthy that the two eyes were not equally affected with loss of mobility; this seems to strengthen the probability that there is a nerve centre governing the movements of each eye. On this point additional evidence is seen in cases where nystagmus affects the two eyes unequally; also in the independent movements of either eye seen in infants in deep sleep, and in adults in coma from chloroform.[1] Again, the greater limitation of movements in the horizontal, as compared with the vertical plane, seems to give evidence of the greater paralysis of the nerve centre governing the horizontal movements, as compared with the degree of paralysis of the centre governing vertical movements. In the normal condition horizontal movements of the eyes are probably the most frequent, and these movements were the most paralysed.

[1] See paper on "Loss of Associated Movements of the Eyes under Chloroform and in Disease," 'Brit. Med. Journal,' March 10th, 1877.

ON THE

ENDEMIC HÆMATURIA OF THE SOUTH-EAST COAST OF AFRICA.

BY THE LATE

DR. VASY LYLE.

WITH AN INTRODUCTION

BY

JOHN HARLEY, M.D. LOND.,

LECTURER ON GENERAL ANATOMY AND PHYSIOLOGY AT, AND PHYSICIAN TO, ST. THOMAS'S HOSPITAL.

COMMUNICATED BY DR. JOHN HARLEY.

(Received March 9th—Read November 14th, 1882.)

INTRODUCTION.

IN my last communication on this subject, printed in the fifty-fourth volume of the Society's 'Transactions,' I referred to Dr. Vasy Lyle, of Durban, as an observer of the endemic hæmaturia from whom we might expect the fullest information concerning its origin and development.

Unfortunately, death has deprived science of this boon, and, what is very remarkable, Dr. Lyle died of hydatid disease of the liver. This event happened nearly three years ago at Prætoria. After practising eight years at

Durban, Dr. Lyle was induced to accompany Sir T. Shepstone to the Transvaal as one of his personal staff. He was present at the annexation of that territory, and, on the formation of a Government staff, accepted the post of Minister of Education.

Since 1864, when I first drew the attention of this Society to the parasitic origin of the hæmaturia of South Africa, Dr. Lyle had taken the most active interest in the elucidation of the disease, and up to the time of his death was making observations and collecting information from every available source respecting its origin and spread. During the whole of this time I have been in frequent communication with Dr. Lyle, and I know that it was his intention to offer the results of his observations and inquiries to this Society. It is some little satisfaction to me that in his absence I am instrumental in doing this. For the means, I am indebted to Dr. Lyle's brother, Dr. Robert Vasy Ash, of the Army Medical Department.

In placing Dr. Lyle's MS. in my hands, Dr. Ash writes to me as follows:—"Among my late brother's letters I found many of yours, and his own notes on the *Bilharzia hæmaturia;* they were evidently intended for you. I am sorry to say that all his microscopical drawings from life are lost." On looking through the MS. I find the information so carefully and systematically arranged that I have not thought it necessary to make any alteration, and I have no doubt that the form in which the Society will receive the communication is exactly that in which Dr. Lyle intended to offer it to us, and I feel sure that the Society, while it appreciates the labours of which the present communication is the fruit, will share in the deep regret which I feel that so able an observer as Dr. Lyle has not been permitted to complete an inquiry for which his extensive knowledge of the diseases of South Africa and his scientific acquirements so eminently qualified him.

Bilharzia hæmatobium. Physical Characters of Infested Country. Extent of Country Infested.

Hæmaturia associated with the presence in the urine of the ova of *B. hæmatobium* has been ascertained by Bilharz and by Griesinger to prevail among the people inhabiting the valley of the Lower Nile, and Dr. John Harley has shown that in the Cape Colony a similar or very kindred helminthiasis exists; my experience proves its existence among the peoples inhabiting Natal and the neighbouring Zulu country to the north. Southward in the Amaponda country I have been unable to determine its existence; the only case coming under my observation being that of a native Kaffir catechist who had contracted the disease in Natal on the Umhloti River. From him and from the European missionary under whom he worked, I have since learned that the disease is unknown among the Amaponda Kaffirs; the district, however, to which their inquiries had been limited is not on the coast but in the uplands.

I have not yet ascertained if this peculiar helminthiasis prevails in the coast country between the latitudes of St. Lucia Bay south, and Egypt north, of the equator; I trust before long to be in a position to supply information on this point, but I suspect it will yet be proved that the *Bilharzia*, whether of one or more species, infests the whole eastern littoral of Africa, from the Nile delta to the Cape of Good Hope.

Of the West Coast of Africa I know nothing personally, but inquiries I have made of those who have for many years together lived in the tropical parts, go to prove that the disease is there altogether unknown.

Inland I have ascertained that hæmaturia prevails midway between Maritzburg and Durban in the neighbourhood of a stream called "Sterk spruit,"[1] and in the town of Maritzburg, situated on the second terrace rising above

[1] See Dr. Harley's second communication, 'Med.-Chir. Trans.,' vol. lii, p. 385.

the sea line and about fifty miles from the coast. Beyond this point I am as yet unable to ascertain the existence of endemic hæmaturia, although I have made inquiry; and particularly as regards the elevated plains beyond the Kahlamba Mountains, the Orange River, and the Transvaal Republics.

In the colony of Natal the *B. hæmatobium* is too common, but the country principally infested is on the coast where the rivers for the most part are sluggish and their courses obstructed with vegetation; in this district, however, a certain degree of immunity is observed in people living on the banks of streams flowing rapidly over a rocky bottom.

Since writing the above I have continued to pursue my investigations on this subject, and with some success, inasmuch as I am now able to assert—not as a mere idea, but as a fact all but completely proved—that the *B. hæmatobium* infests the whole eastern littoral of Africa from Egypt to the Cape, and that the entozoon found amongst the people of the Nile Valley is identical with that found in South Africa.

That the entozoon exists in the neighbourhood of St. Lucia Bay I have ascertained from a case coming under my own observation; my first inquiry was consequently directed to ascertain if in the vicinity of Delagoa Bay cases of this peculiar hæmaturia are common; unfortunately my inquiry had to be addressed to a layman, Señor de Paiva Raposa, the Government secretary there. This gentleman replied to my letter with promptitude, but his evidence is entirely negative, for he says (under date 13th February, 1873), "I beg to tell you that the worm which affects people on the districts of Eastern Africa is not known in the country around Delagoa Bay." Indirectly I have learnt that this information is incorrect, that the district around Delagoa Bay is infested, but I give the statement of Señor de Paiva Raposa as I received it.

My next inquiry was addressed to a practitioner long resident at Mozambique; from him I have as yet received no reply.

With Dr. Kirk of Zanzibar I have been more fortunate and from him I have obtained valuable information. By letter dated 7th October, 1872, he writes: "Regarding your inquiries, I can state that between the years 1858-63, I travelled up and down the Coast between this and the Zambesi mouths, and lived for years in the interior, passing all the Zambesi to above the Victoria falls and on the Shire to Lake Nyassa. I then frequently saw the disease which I now know to be caused by the *B. hæmatobium*. Its symptoms were to me new and I classed it as an endemic form of hæmaturia, different from any then described. . . . I knew nothing of the pathology of this peculiar disease until, in London, Sir T. Watson called my attention to Dr. Harley's paper in the 'Medico-Chirurgical Transactions.'[1] I then at once recognised the disease I had seen so much of.

"It was peculiarly common on the lower part of the Zambesi at Shorganga and Senna, where the natives name it '*Tanda moropa*' or 'the passing of blood.' To them this term signifies a specific disease.

"I treated several cases and found gallic acid gave relief from the symptoms but saw no cure, although, at the same time, I saw no case fatal. The disease exists though less common in south latitude 18°. I have been now upwards of six years stationed at Zanzibar, but am now engaged as the British agent. My opportunities of observation are therefore very limited, but I have no doubt from what natives tell me that the disease exists on the mainland. It seems to me to follow rivers and low marshy lands and to be absent from mountains, but it must be kept in mind that a traveller has few opportunities of getting information on such matters. I should not have known the disease existed on the lower Zambezi, had not the servants who followed me been from that part."

[1] Vol. xlvii, 1864.

Dr. Kirk's letter proves the existence of the disease on the mainland of eastern Africa, at a point which may be said to be midway between Egypt and the Cape, but indirectly I have ascertained that it also prevails in the country around Inhamban. I have no doubt more accurate observations than those as yet available will prove its existence wherever, on the Eastern littoral of Africa, shallow, sluggish-flowing streams traverse an inhabited country.

Up to the present time my observations go to prove that the interior plateau of South Africa is free from this particular form of helminthiasis. I am informed by Mr. Wilson, who was a companion of Livingstone in his earlier travels, and who has resided twenty-two years in the interior, about Lake Ngami in Damaraland, Namaqualand, and north-west of Lake Ngami on the Teoghe river where there is much swamp and the water-courses are impeded with vegetation, and also in the upper part of the Zambesi above the Victoria falls, that he never heard of the disease. Five years also he passed in the district of Walwich Bay, and there the disease was unknown. My informant added: "Livingstone is still remembered by the people of the districts he travelled through, but always by a name meaning 'the doctor.' As I was with him during these journeyings I am looked upon as in some sort a doctor to, and am continually applied to for medicines. I speak the languages of the natives, and am certain if the disease you describe prevailed it would have come under my notice." Another traveller (Mr. Higgs), who had also passed many years in the interior, entirely corroborates Mr. Wilson's statements.

In support of the opinions already advanced respecting the prevalence of the disease in Natal, I quote from a letter received from the Rev. John Allsopp, a missionary, resident here for many years, a competent observer, and cautious in drawing conclusions. He says: "I find it (hæmaturia) almost everywhere along the coast, and have heard of some cases as high up in the colony as Mooi river. It is more common with boys than girls; I account for it

thus: the boys (speaking of the 'natives') are out all day herding cattle, they get together in threes and fours, play in and about the smaller streams, and of course drink a good deal of the water; the girls bathe but seldom, and when they do it is generally in the larger rivers." In respect to Europeans: "I have not met with a single case in youths confined to town life and accustomed to drink rain-water. I am persuaded that the water of small streams running through vegetation and over stones of varied kinds is more calculated to produce the disease than the water of rapidly flowing and sandy-bedded rivers."

Mr. G. J. Cato, of Cato Manor, whose acquaintance with the natives of Natal, their language, and their habits, makes him a good observer, writes: "I am of your opinion that both sexes are subject to the disease, and certainly those are most so who use water from marshes, pools, rivers with shaly beds having marshy banks or sources; on the contrary, the inhabitants of the valleys of sandy-bedded rivers with the same surroundings seem free.

Mr. Cato criticising a remark of Dr. Dunsterville, of Port Elizabeth (quoted in Dr. John Harley's second communication, 'Med.-Chir. Trans.,' vol. lii, p. 380), to the effect that "they have not nearly so much of the disease as formerly," further remarks: "I account for this as follows:—Port Elizabeth, or rather the town, is situated at the base of a hill, and formerly water was very scarce there, for wells had to be dug wherever a damp place could be found, and I have known water from such pits—'good water' it was called—retailed at prices varying from a farthing to a halfpenny the bucket, or at a fixed sum the month; but now nearly everyone, at least all who can afford it, drink rain water, which is collected and stored in tanks."

I trust you will pardon me for being thus particular—verbose I may say—in laying this evidence before you, for I wish you to form your own conclusions, not simply to adopt mine. Beyond this, it appears to me, that it is only after rightly appreciating the causes of a disease and

its peculiarities of occurrence that we can recommend a sensible prophylaxis.

Description of the Entozoon.

The description of the *Bilharzia hæmatobium* given by Cobbold ('Entozoa,' p. 197) appears to me correct, and agrees, in all but minor details, with the results of my investigations.

The following are the facts observed by me:

November 21st, 1872, I obtained the bladder of a patient who had suffered from the endemic hæmaturia, from the Durban Hospital.

The internal surface of the viscus was healthy near the neck, but was crossed diagonally by fungous-looking growths from near the cervix to the fundus. One protuberance was pedunculated and as large as a hazel-nut. The abnormal mucous membrane presented superficial ulcerations, and there were many ova, some granular and some fully developed, embedded in the adherent mucus.

A black line was observed and dissected out. It proved to be a *female* Bilharzia. It was one inch long and filiform. Ova were contained in the hinder part of the body, and these were granular; the pointed end was not always in one direction, but generally opposite the outlet; they were arranged mostly in single file. The intestine divides into four gradually diminishing canals, which soon reunite to form a broad central sacculated tube, which extends down to the middle of the body, and terminates in a *cul-de-sac*. The characteristic spine of the egg was always terminal.

I obtained the posterior fragment of a *male* specimen, December 5th, 1872. The body was roughly tuberculated and tapered to a conical tail, reminding one of the crocodile. The alimentary canal presented anastomoses, and the gynæcophoric canal was continued to the tail.

I have elsewhere mentioned that I have not yet met

with a case where death could be directly attributed to the presence of *B. hæmatobium* in the human system. This appeared to me singular so long as I accepted as correct the statements of Cobbold, Bilharz, Griesinger, and others, that the habitat of the worm is in the "portal system of blood-vessels," "in the veins of the mesentery, bladder, and other parts;" for, I could never understand why the worm or its products should not be occasionally carried along the blood current, and eventually by obstructing some of the smaller vessels give rise to serious if not fatal results. Nor will I deny the possibility of the occurrence of such accidents; I can only assert that none such have as yet fallen under my observation. But since I have ascertained that the worm resides in the cellular tissue surrounding the bladder, and principally in that between the bladder and rectum, I can understand why the *Bilharzia* is usually so harmless to life. In time and after increase it may migrate and invade the ureters, the kidneys, and the blood-vessels, but as yet my dissections have not verified this opinion.

Symptoms and Progress of the Disease, with Illustrative Cases.

There do not appear to be any symptoms marking the introduction of the *Bilharzia hæmatobium* into the human body; at least up to the present time nothing of the kind has been recognised. The earliest indication that the individual is the host of this entozoon occurs when the worm is sufficiently mature to shed fertilised ova, and at this time the patient begins to pass urine the last few drops of which are mixed with blood. An examination of this bloody urine with the microscope has, even in the earliest stages of the disease coming under my observation, revealed the presence of many fertilised ova of the *B. hæmatobium*. The detection of these ova is most important, giving certainty to the diagnosis; hæmaturia may arise from many causes, but

its association with the eggs of this entozoon removes all question as to cause in any given case. There are characters about the hæmaturia itself sufficient to call the attention of an experienced observer to the real nature of the case; it presents this peculiarity—only the last few drops of urine are mixed with blood; the bulk of the urine voided is perfectly free from all unnatural colour. The explanation of this peculiarity rests with the fact that the last part of the urine is expelled mainly by the action of the muscular coat of the bladder; any minute ulcerations or tunnels made by the worms are thus subjected to a disturbing influence and rendered liable to bleed.

In this, the earlier stage of the disease, there is singularly little general distress. Some patients complain of lumbar or of perineal pain, described as a dull aching sensation, occurring only occasionally and, according to my observation, noticeable in but few cases.

The quantity of blood lost in the earlier stages is small, amounting only to a few drops at each act of micturition; in some cases this may never materially increase, it may lessen, become irregular in occurrence, and eventually disappear so entirely that blood is not to be detected in the urine with the unaided eye; the patient then flatters himself he has got rid of the parasite; he is, however, mistaken, for long after the subsidence of all visible bleeding the eggs of the entozoon may be detected in the urine, and remote consequences arise. Dr. John Harley was the first to call attention to this important fact in his paper read before the Royal Medical and Chirurgical Society,[1] and I am able to corroborate his statement; as an example I call attention to the following case.

Case 1.—April 4th, 1872.—H. F—, æt. 20, planter's son living in the valley of the lower Umhloti, Co. Victoria.

Within the last three months he has had three attacks of lumbar pain, affecting principally the left side, but occa-

[1] First communication, vol. xlvii, p. 65—7.

sionally the right; the pain strikes toward the genitals and is accompanied with faintness and nausea. He says he does not pass blood in his urine, but years ago used to do so with the last few drops; at this time he was eight years old. This symptom soon disappeared and has never returned.

Examination of urine.—Urine amber coloured, clear, but with minute floating flakes (of mucus). Sp. gr. 1023. Rendered turbid by heat, but the deposit dissolves with effervescence on the addition of hydrochloric acid.

One of the flakes of mucus was examined by microscope and observed to contain many ova of *B. hæmatobium*, some mature others immature, containing sarcode granules. The ova were elongate-ovate with the characteristic pointed end; there were also noticed epithelial cells from the bladder full of oil globules, many blood discs and mucous cells.

Subsequently (September 21st, 1872) I had an opportunity of examining this patient during one of his nephritic attacks; the symptoms were such as already described, but very severe, the prostration was extreme. The symptoms yielded rapidly to anodynes and warm baths.

It is not always that so little disturbance as that described follows the introduction of the entozoon; occasionally amongst all classes, and I may say, very frequently, among the weakly Indian immigrants, the bleeding goes on increasing until the quantity of blood lost at each act of micturition is considerable, and voided not only mixed with the urine but in clots. As may be expected, in such cases there is anæmia and debility, and at times much difficulty in passing urine from the mechanical obstruction offered by the clots. Now, the perineal distress and the lumbar pain become marked and a true vesical catarrh occasionally sets in, a complication not easily cured, as its cause is to a great extent beyond the reach of our remedies. Grave as is this picture I must, however, state that I have not yet met with a fatal case of *B. hæmatobium*, whether arising directly or indirectly from the presence

of the entozoon. This latter statement I must, however, qualify, for I do not yet know to what extent the parasite may be a factor in the production of suppurative inflammation of the liver, the worm or its ova passing by the portal circulation to the liver, and thus affording a point of origin for inflammatory action.

Dr. Cobbold ('Entozoa,' p. 35) obtained a male specimen of the *B. hæmatobium* from the portal vein of an African monkey, *Cercopithecus fuliginosus*, which died at the Zoological Society's menagerie. I have myself observed great enlargement of both liver and spleen to occur in patients harbouring the *B. hæmatobium*.

Apart from what has now been mentioned, it seems to me probable that in the future very serious results will be traced to the presence of the *B. hæmatobium* in the human system, and the accidental entrance of itself or its products into the circulation.

Incidentally I have already mentioned the occurrence of nephritic colic in patients who have for a long time been the victims of the *Bilharzia*. These attacks are of such common occurrence in old-standing cases that I have come to look upon them as marking a third stage: a consequence, if not invariable, at least very frequent. The attacks seem to me to be sometimes caused by the passing of a clot of blood, more often by renal calculus, sometimes phosphatic, but more frequently oxalate of lime. The occurrence of crystals of oxalate of lime in urine is, it seems to me, more common in Natal than in Europe. I am therefore disposed to think the more frequent occurrence of this particular form of calculus associated with a history of a particular helminthiasis is accidental, and not immediately depending on the presence of the entozoon; certainly, reasoning from pathological conditions, it is the phosphatic not the oxalic calculus one would expect to find as a consequence of the parasitism under discussion.

As is shown in another part of this paper, the parts of the human body most liable to the invasion of the parasite

are the bladder and the structures connected with it; the earlier symptoms of the helminthiasis are consequently referable to this viscus. As the disease progresses these symptoms increase in degree, and complications arising out of continued local irritation may occur; lastly, and as a remote consequence, renal irritation may set in, and as a result calculi are formed.

How it is that the kidneys are eventually involved in the spread of this disease I cannot as yet tell; it may be that irritation spreads to the pelvis of the kidney by continuity, or it may be that the entozoon actually travels there along the ureter or burrows its way along the surrounding cellular tissue. This with many other questions respecting the propagation and habits of the *Bilharzia* and its remoter effects on the human system remain for future investigation.

Case 2.—1870, June 18th. Maquaka, a lively, well-nourished Kaffir girl, æt. 7, came under observation in consequence of a large wart affecting the left of the labia majora. During examination I ascertained that in voiding urine the last few drops were invariably tinged with blood; this symptom had been noticed for one month, no other deviations from health observed. She drinks water drawn from a pit in the soil, not in the rock, and is accustomed to bathe in the Umsindusi, an affluent of the Umhloti river.

Examined under the microscope the urine was observed to contain blood and many ova of the *B. hæmatobium*. Santonine was prescribed in doses of one grain and a half every morning.

September 6th.—She again presented herself; the drug had been irregularly taken, and some benefit appeared to have resulted, as the bleeding was reported to be less.

28th.—I was informed by letter there was a marked improvement, the child only occasionally passing blood. (No later information.)

1870, June 18th.—The Kaffir girl whose case is shortly

related above was accompanied by her father, a powerful-looking fellow, apparently thirty years of age, and by a relative who, having visited England, speaks our language well, and on this occasion acted as my interpreter. The father said, "I know that men, girls, and boys suffer from the disease we are talking about in the district where I live; women may suffer from it also, but of course I do not know. I have suffered from it since my boyhood; I still do so, and am always worse if I walk a long journey. Our people have medicines which they give for this complaint, but I have never heard that anybody was made better by taking them. When first I observed I passed blood with my urine I was living on the Umfolosi (a river flowing into St. Lucia Bay). The people of my village used to draw water from the Umfolosi, and I used to bathe in it. I am aware that a great many people in Zulu, as well as in Natal, suffer from this disease. I know too that calves suffer from a similar disease, never oxen or cows. I never saw dogs suffer from anything like it."

Tye, my interpreter (a Kaffir), says:—"I too have had the disease; I first noticed it when I was about fourteen years of age, and whilst I was living near the Tongaati river. The water we used was drawn from a hole in the ground. We bathed in the Tongaati. The disease lasted about three years, and then went away after living a time at Durban, where I always drank rain water. Many people at the Tongaati suffer as I did; for my part I have now been quite well eight years. I have seen calves suffer from a similar disease."

Causes of Bilharzia hæmatobium.

The manner in which the entozoon is introduced into the body of the host has not yet been demonstrated, everything I have read on the subject is conjectural, and I regret to say in the following remarks I can offer nothing different.

My attempts to rear the larvæ of *Bilharzia* have hitherto been unsuccessful: a result similar to that attained in the more experienced hands of Dr. Cobbold and Dr. John Harley.

I have never had any difficulty in obtaining the well-developed ciliated animalcule, and have observed the various shapes it assumes under different circumstances, but know of nothing which points the way to a recognition of the next form in the metamorphosis.

According to Cobbold ('Entozoa,' p. 199) it appears Griesinger conjectures "that the young of *Bilharzia* exist in the waters of the Nile, in the fishes which therein abound, or even in bread, grain, or fruit." That they do exist in the waters of infested countries I have no doubt, but from the facts I am about to relate I have equally no doubt they are not in Natal introduced into the human system in the manner supposed; bread, grain, and fruit are consumed equally by the population of town and country, whether European, Kaffir, or Indian, yet the population of the town of Durban remains as yet free from the pest. The Kaffirs scrupulously avoid eating fish and yet they are affected. It appears to me one must seek for a more universally employed agent of introduction, and that is afforded in the water used for domestic purposes. In the town of Durban rain-water is used, or water drawn from wells; elsewhere in the colony, with very few exceptions, the nearest river or pond supplies all that is needed.

The above remarks will apply with greater force, as will be illustrated further on, to Cobbold's own suggestion that "certain gasteropod mollusks" are the intermediate bearers and vehicles for introduction.

I confess my own observations lead to nothing definite, they, however, seem to point out the direction our precautions should take to avoid contracting the helminthiasis whilst residing in or travelling through an infested district.

In his papers read before the Royal Medical and Chirurgical Society, Dr. John Harley recognises the possibility

of the disease being contracted by drinking or bathing in the impure waters of infested districts, and my observations go to support this conclusion; but in the last of his published papers which I have seen he goes further, and his attention having been drawn to the existence in Natal of a peculiar ulcerous affection of the skin, locally known as "Natal sores," he suggests that this may be a local phenomenon arising from the reception of the *Bilharzia* in one stage of its growth, and may mark the first stage of its introduction into the system.

My acquaintance with the so-called Natal sores has been extensive, and I soon ascertained that the term is used by the laity to mean any ulcerous affection whatever of the skin. But there is a disease prevailing in the colony, from which immigrants so frequently suffer, to which the term "Natal sores" has been generally attached. This disease seems to me a form of ecthyma, of a chronic nature and for the most part occurring in people just landed, or others who, though not recently arrived, have undergone privations, whether incident to position or to travelling. "A local expression of general debility," is a formula of words suited, in my opinion, to convey the physical condition of the sufferer, a general debility induced by privations on shipboard, or after landing, or in travelling, or by intemperance, or it may be through local debility from overaction of the skin in an almost tropical country; but, as far as I can find, it is in no way connected with the hæmaturia endemic to South Africa. It is notorious that immigrants recently arrived are more frequent sufferers than old residents; so much so is this the case that it is here looked upon as in some measure an acclimatizing process, erroneously so I think. These being the facts relating to this affection, it can hardly be concluded that the occurrence of Natal sores and of hæmaturia from the presence of *B. hæmatobium* have any connection; indeed, I have been unable in any one instance to connect the two diseases, although my inquiries have been specially directed to this point since I first read Dr.

Harley's paper. In corroboration of these statements, I should mention that Natal sores are common among the different peoples inhabiting this colony, and equally among the populations, urban and rural, whilst hæmaturia, as is elsewhere shown, is not.

During the many years I have been engaged in practice in Natal, and notwithstanding diligent inquiry, I have been unable to meet with a case of hæmaturia arising from the presence of *B. hæmatobium* in a person who has principally resided in the town of Durban. One case has come under my observation occurring in a man living in the inland town of Maritzburg, where this peculiar form of hæmaturia is said to be prevalent. In the case now mentioned the first signs of the disease were noticed at ten years of age, up to which time my patient had never left the vicinity of the camp and town.

The disease is, however, by no means rare among people residing in the rural districts of the infected country, and as far as my observations go, those are most frequently affected who from occupation or carelessness are most likely to use impure water. Thus adult European women, and particularly those of the better classes, are but little affected. I have not yet met with a case occurring in a *lady* of European birth who had attained puberty before coming to this country.

Men of European birth are affected, but they are those who are hunters or travellers, or overseers of labour—the latter accustomed to be for the whole day in the field with their men—and all from the force of circumstances likely at times to have to use impure water, both for drinking and for bathing.

Once a series of cases came to my notice corroborative of the foregoing statements. I was consulted by a gentleman living in the neighbourhood of this town, in a district where rainwater is used for all house purposes, and requested to ascertain the cause of the hæmaturia with which two of his boys were afflicted. Having satisfied myself that in both cases the bloody urine was asso-

ciated with the presence of many of the eggs of the *B. hæmatobium* I paid a visit to the house to inquire into the circumstances of the origin of the *hæmaturia;* on inquiry I was told that nothing but rainwater was used for cooking, washing, or drinking. I remarked that the circumstances of the origin of the parasiticism upset all my previous conclusions, stating at once what they had been. The mother of the boys then told me that she had first noticed the hæmaturia a short time after she had permitted them to go with the children of S— to bathe in a rivulet flowing through a swampy valley at the foot of the hill. Not long afterwards I was consulted about the cases of the other boy bathers, and ascertained that they too suffered from hæmaturia and passed with the blood the eggs of the *B. hæmatobium.*

Kaffirs of both sexes are affected, and they recognise the characters of the districts where the disease is engendered, and are influenced thereby in choosing the site of a settlement; nor, curious as it may seem, are they altogether ignorant of the immediate cause of the bleeding. Their habits are such—on the supposition that the parasite is introduced into the human body through an intermediate bearer—as should protect them, for they scrupulously avoid eating fish or "gasteropod mollusks" or frogs, nay, a pure-blooded Zulu will eat nothing which in his way he cannot understand; he will rather starve. He will not eat what he considers grotesque, whether animal or vegetable; and so it is that in the whole colony of Natal there is only one fish-eating class, despised too for it by the others; it inhabits a sea-bordered corner into which it was driven during the wars which prevailed before European rule was established. But all these Kaffirs drink where they can when they are thirsty, and bathe too if a hot skin prompts.

The manner in which a Kaffir drinks from a stream is peculiar; he flings the water with his hand to his mouth, and tells you, if you ask the reason, it is to avoid introducing small floating things.

Natives of India—immigrants to this country—are affected by the parasite, and it appears to me, more frequently than either Europeans or Kaffirs; their methods of cooking protect them from the too common *tænia* of South Africa, but they seem to me to suffer from the *B. hæmatobium* more generally and more severely than any race inhabiting Natal, neither age nor sex appearing to afford protection. But if there is any truth in my observations respecting the localities in which the entozoon most prevails, Indian immigrants more than any other class in Natal should be subject to its ravages, for they are employed for the most part on sugar and on coffee estates. These are situated in the littoral and infested districts, where the streams are frequently sluggish, flowing through swampy valleys, and hold much organic matter in suspension. To afford facilities for bathing and for procuring water for cooking purposes and for drinking, the cottages—hovels I ought to call them—of the labourers are placed as near as convenient to a watercourse or, it may be, to a marshy pool; the available water, previously impure from locality and causes which for convenience may be called natural, is frequently rendered still more impure by the intermixture of drainage from the sugar manufacture or coffee works. I confess the suspicion that this constantly increasing impurity of the water has an influence in increasing the frequency of diseases associated with *B. hæmatobium* has arisen in my mind. I am intimately acquainted with one locality where the aspect of the river valleys on the estate is such as I am accustomed to find where the disease prevails. I resided there for many years, bathed in the waters of the streamlets, and with my family and servants drank of them; we had no other water—and yet during the eight years I lived there I found but very few instances of the disease, and its origin was for the most part to be traced to other localities. No instance of the disease has occurred in my own family. Subsequently a large sugar mill and a distillery were established on the estate, the drainage from which necessarily flowed into the

river above the point where the coolie village was established. In time this estate became as notorious as others for the occurrence of *B. hæmatobium* among its people, and is now one of the worst infested.

I have already mentioned the reputed difference between the towns of Maritzburg and Durban as regards the chances of infection; this difference is I believe real, and its explanation may be found in the fact that at Maritzburg water has been brought into the town and flows through open channels on each side of the principal streets, whilst at Durban, no such facilities for water supply occurring, the inhabitants use rainwater collected from the roofs of their houses and stored in tanks.

When the question of water supply for towns in South-eastern Africa after the European fashion comes to be discussed, an important element in the inquiry will be, Can river water be used with safety?

A CONTRIBUTION

TO THE

PATHOLOGY OF DIPHTHERITIC PARALYSIS.

BY

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Received December 7th, 1882—Read January 9th, 1883.

The clinical features of diphtheritic paralysis are now well known. Some difference of opinion, it is true, still prevails concerning the frequency of its occurrence and the danger which it entails. But there is a more or less general agreement as to the clinical aspects of the disease.

Much uncertainty, however, has from the first attached to its pathology. It is only in more or less recent times that there has been any record of definite facts bearing on the question. Consequently the literature relating to the pathological anatomy of diphtheritic paralysis is somewhat limited.

Charcot and Vulpian,[1] as far back as 1862, described fatty and granular degeneration of the palatine nerves and their corresponding muscles. In this case the paralysis of

[1] 'Compte rendu de la Soc. de Biol.,' 1862.

the palate developed eight or nine days after the angina appeared.

There was no paralysis of other parts, and no mention is made of any microscopic examination of the spinal cord.

A few years later Lorain and Lépine[1] stated that they had observed changes similar to those described by the last authors but gave no further details.

Liouville[2] found similar degeneration of the phrenic nerves in one case.

Buhl,[3] in an article entitled "Einiges über Diphtherie," related the results of an autopsy of a case of diphtheria in which he found marked lesions of the nervous system. The examination revealed extravasations of blood in almost every organ, and also in the cerebral pia mater and cortex. Infarcts were found in the white matter of the brain, in the central ganglia, crura cerebri, pons, cerebellum, medulla, and spinal cord. The spinal ganglia, and the anterior and posterior roots at their points of junction, were swollen. The nerve sheath and ganglia being infiltrated with nuclear bodies, which Buhl considered to be characteristic of the false membranes of diphtheria. These appearances were most evident in the lumbar region. No mention is made of any affection of the spinal cord itself. The absence of any clinical history of paralysis forbids any stress being laid on this interesting case.

Hermann Weber, "Ueber Lähmungen nach Diphtherie,')[4] gives an account of paralysis in an adult. Four weeks after the sorethroat began this patient had recovered completely. But two weeks later he was seized with paralysis of sensation and movement, first in the legs, then in the arms, and lastly, he had symptoms of paralysis of the sphincter vesicæ. The paralysis came on after the patient had walked a great distance and had got wet through.

[1] Art. "Diphthérie," 'Nouv. Dict. de Med. et Chir. pratique,' 1869.

[2] Quoted by Bailly, 'Thèse de Paris,' 1872.

[3] 'Zeitschrift f. Biol.,' 1867.

[4] 'Virch. Arch.,' xxv, p. 130.

Death occurred twelve days after the paralysis began and was preceded by great dyspnœa. The autopsy revealed fatty degeneration of the heart, œdema of the lungs, congestion of the kidneys; no naked-eye or microscopic change in the nervous system.

Leyden[1] found in one case the appearances of a "neuritis migrans" ascending towards the nerve centres.

Oertel[2] in one case met with multiplication of nuclei in the spinal cord, especially in the anterior cornua, and hæmorrhagic patches in the cord and pia mater.

Vulpian[3] in 1870 published the results of an examination of three fatal cases but without any clinical details. In two of these he found what he calls "rarefaction" of the connective tissue in the outer and posterior parts of the anterior horns, and alterations in the motor nerve-cells. The affected cells were globular in shape, their contents had become homogeneous, and the nucleus was obscured. There was also a slight increase in the number of nuclei in the spinal cord. Vulpian did not, however, attach any precise importance to these changes.

Pierret[4] a few years later described a case of paralysis in an adult, in which he found disseminated patches of meningitis with perineuritis of the adjacent spinal roots.

It was reserved for Dejerine[5] to bring forward the first definite facts in favour of diphtheritic paralysis being due to a degeneration of the motor nerve-cells of the spinal cord.

In three cases of his own and in two others to which he had access, he found constant lesions of an inflammatory nature in the anterior cornua, with consecutive neuritis of the corresponding anterior spinal roots. The posterior roots in each case were unaffected. The lesions of the anterior bones were both parenchymatous and inter-

[1] "Ueber Reflex Lähmungen," 'Sammlung Klin. Vorträge v. Volkmann.'
[2] 'Deutsches Arch. f. Klin. Med.' viii, 1871.
[3] 'Malad. d. Syst. Nerv.,' 1870.
[4] 'Comptes rendus de la Soc. de Biol.,' 1876.
[5] 'Arch. de Phys. Norm. et Path.,' tome v, p. 107, 1878.

stitial. These nerve-cells were swollen in some instances, shrunken in others. The affected cells were indistinct, had lost their processes, and were globular in shape.

The number of nerve-cells in the anterior cornua was diminished. These changes were most marked in certain parts of the spinal cord.

The neuroglia showed multiplication of its nuclei and took up carmine very readily, an evidence of irritation as Dejerine thinks.

The small vessels were distended with blood and dilated. Hæmorrhages due to rupture of vessels were found in the anterior cornua, together with perivascular collections of small cells.

Around the central canal he found cell infiltration of an inflammatory nature. This affection of the spinal cord is regarded by Dejerine as a "subacute tephromyelitis," the neuritis of the anterior spinal roots being secondary to the spinal lesion.

Dr. Abercrombie at the last International Medical Congress in London gave the results of a microscopical examination of the spinal cord in seven cases of diphtheritic paralysis. In all of these cases he found evidences of a more or less advanced degeneration affecting the motor cells of the anterior bones in different parts of the cord.

These changes resembled closely those described by Dejerine, but though the small vessels were filled with blood no hæmorrhage was seen. There was a doubtful increase in the nuclei of the neuroglia, but no stress is laid by Dr. Abercrombie on this point. This author also considers the spinal lesion to be due to a "subacute myelitis."

It will be seen from this short historical sketch that although Vulpian was the first to recognise changes in the motor cells of the spinal cord, he did not attach the importance to this degeneration for which Dejerine and Dr. Abercrombie have since contended.

I shall now proceed to give a short description of a fatal case of diphtheritic paralysis that came under my

own observation and in which I had the opportunity of examining the nerve centres.

In the present state of the pathology of this affection every fatal case is of the greatest importance.

This must be my excuse for bringing forward an account of a single case.

Ernest H—, æt. 7, was brought to me by his father at the out-patient department of the Brompton Hospital with the following history:

Two months ago the boy had diphtheria, ever since he had the sorethroat his voice has been nasal, and food has sometimes come through his nose. He has been very weak all along, and never regained his strength properly. Two weeks ago he began to cough and since that time he has had some shortness of breath.

Present condition.—The patient is a pale, thin boy. Voice distinctly nasal. Seems very weak and walks in a tottering way. Muscular power of arms small, grasp of hands very feeble. Legs weak but less so than arms. Knee-jerk feeble on both sides. No distinct loss of cutaneous sensation. Micturition and defæcation unaffected. Tongue protruded straight, movements rather tremulous. Pupils contract with accommodation and react to light. Soft palate hangs motionless. No distinct action of the intercostal muscles. Breathing diaphragmatic, rapid. Chest: physical signs of general bronchitis.

As the intercostal paralysis was evidently a source of danger, I advised the father to try to procure admission for him at once into a general hospital, and ordered him an expectorant for his bronchitis. Three days later the father came and told me that the child was dead. He had been unable to get him admitted at the hospital where he applied, and had therefore kept him at home.

His breathing became gradually shorter and he died rather suddenly, before a doctor could be summoned. Death occurred two days after I saw the patient.

I regret that the clinical account of the case is not more complete, but on the only occasion in which I saw

the patient his general condition necessitated a somewhat hurried examination.

I proceeded to make the autopsy twenty hours after death. But when I had removed the brain and spinal cord the father interfered and refused to allow me to complete the examination.

The brain, spinal cord, and the membranes presented no alteration to the naked eye. The spinal cord, medulla, and pons were placed in weak alcohol for twelve hours, and were then transferred to a 2-per cent. solution of bichromate of potassium for three weeks. Hardening was subsequently completed with alcohol.

The pons, medulla, and spinal cord throughout were examined systematically. Sections were cut with the freezing microtome, and after staining with hæmatoxylin or picro-carmine were mounted in Canada balsam.

Results of microscopical examination.—Certain slight changes of a similar character were observed throughout the parts examined. These consisted in an accumulation of leucocytes in the smallest veins and in the capillaries. In a few instances the small veins were seen to be crammed with red corpuscles, especially in the pons and medulla. There was also distension of the capillaries and smallest veins in places. Collections of small cells, for the most part contained in capillaries, were observed around the cerebral canal throughout the cord.

Besides this vascular affection, definite changes were found in the grey matter. These changes concerned exclusively the anterior cornua, and were most marked in the dorsal region, less so in the cervical, and least of all in the lumbar division of the cord. The medulla and pons showed no alteration beyond the capillary and venous distension already alluded to.

The changes presented by the anterior cornua consisted in atrophy and alterations in the shape and appearance of their nerve-cells. As these changes attained their greatest development in the dorsal region a description of sections taken from this part will suffice for the cervical and

lumbar regions also. The changes in the latter parts differ from those in the dorsal cord only in degree.

Changes in the nerve-cells.—In many sections of the dorsal region examination with a low power shows numerical atrophy of the anterior nerve-cells, which in some cases is more marked on one side, in others is equally well marked on both sides. In such cases the sharp outline of the cells of Clarke's posterior vesicular column and of the tractus intermedio-lateralis contrasts strongly with the ill-defined appearance of the anterior nerve-cells.

However, on examining the anterior horns with a power of $\times 300$ in such cases I have invariably found evidence of the presence of more nerve-cells than one would expect from the previous examination.

These cells have undergone changes that readily explain the difficulty found in recognising their presence with a low power.

In the same anterior cornua, side by side with large, sharply-outlined, multipolar nerve-cells with branching processes, are seen small indistinct cells which have lost their processes. These cells have also a tendency to assume a more or less globular form in some instances, in other cases they are irregularly shaped.

As a rule, the cell protoplasm has become very pale and indistinct, and it is often somewhat difficult to distinguish such cells from the surrounding neuroglia. Vacuoles are found in some of these pale indistinct cells, and the nucleus is generally indistinct or invisible. The globular cells, on the contrary, are often more granular than natural, and stand out sharply.

These granular cells often show a well-marked nucleus. In nearly all these degenerate cells of both types careful focussing shows traces of the original cell processes. In no case did I see any swelling of the nerve-cells. The changes that I observed all tended towards atrophy.

In a few places the neuroglia is coarsely granular. In the neighbourhood of these granular patches are seen degenerate nerve-cells, which are partially encircled by a

somewhat crescentic, coarsely granular mass, resembling the surrounding neuroglia from which it is probably derived. These changes, however, have only a very limited distribution.

With this slight exception the neuroglia shows no change either in its intimate structure or in the number of its nuclei in any part of the spinal cord.

The affection of the motor cells, on the contrary, was found in almost every section from the dorsal region, and was not confined to any particular cell group of the anterior horns. The nerve-cells of the tractus intermedio-lateralis are unaffected in the dorsal region, though in the cervical segment similar but less marked alterations than those above described are seen in the cells of this group also.

The anterior nerve-cells of the cervical cord show exactly similar changes, but a relatively smaller number of cells is seen to be affected. A fairly large proportion of healthy cells is present in this region. Some sections show only very slight affection of the motor cells.

In the cervical as in the dorsal segment no particular group of nerve-cells is specially attacked.

In the lumbar region almost all the motor cells are quite healthy in appearance, here and there one or two small rather indistinct cells can be seen.

The nerve-cells of the pons and medulla are also quite healthy. No change is visible in the nerve nuclei on the floor of the fourth ventricle.

Drawings are given illustrating the appearances of the affected nerve-cells.

It now remains to estimate the importance of the foregoing changes in the spinal cord. There can be little doubt, I think, that evidence has been given in the present case of a distinct degenerative affection of the motor nerve-cells in special regions. In other words, we have to do with a "polio myelitis anterior."

This polio myelitis is characterised by a somewhat irregular distribution. Healthy and degenerate cells are

seen in one and the same cornu, although in those parts where this affection is most marked the latter decidedly outnumber the former.

This fact to my mind would be enough to negative the hypothesis of a more putrefactive change, if the general appearance of the tissues did not itself sufficiently discredit such an idea. The question as to whether a numerical atrophy of the motor cells is present or not in the affected parts is harder to answer.

This certainly seems to be the case in some sections from the dorsal segments. It will perhaps be objected that in this region the motor cells are least abundant in the normal spinal cord.

This is true as far as it goes, but I think the objection is hardly a sufficient one, as will appear on comparing these sections with those of a normal dorsal region. It may be that owing to their shrunken state the degenerate cells become loosened and so get detached in the various stages of cutting, staining, and mounting the sections. On the other hand, it is not improbable that total disappearance of the motor cells is the last link in the chain of pathological processes of which the degenerative change in the cell protoplasm is the starting point.

A "poliomyelitis" anterior of irregular distribution would sufficiently explain the paralysis that follows diphtheria. Lesions affecting units of a group of motor cells could hardly fail to result in imperfect function of the whole group. And it must be remembered that diphtheritic paralysis is usually rather a weakness or paresis than a complete paralysis.

The varying degree and extent of the poliomyelitis in the different segments of the spinal cord seem to have been accurately represented by the different degree of paralysis in the corresponding muscles. The capillary and venous distension and the accumulation of blood corpuscles in these vessels seems explicable from the fact that the patient died of slow asphyxia. The collections of

cells around the central canal are probably to be explained in the same way.

In any case this last appearance has only a very doubtful significance in the present state of our knowledge.

No change in the motor nuclei of the medulla was found corresponding to the immobility of the palate. This is not to be wondered at when we remember that the patient's voice was said to have been nasal ever since the angina appeared.

Local causes seem accountable for this condition, whether the diphtheritic lesion affected primarily the muscle-fibres or their terminal nerve endings, though the latter supposition seems more likely.

True paralysis is a sequela of diphtheria, and rarely comes on before the second or third week, while it often developes much later. It is difficult to say when the general muscular weakness set in. But the palsy of the intercostal muscles probably commenced six weeks or so after the onset of the disease, about the time that cough and shortness of breath were noticed.

On this hypothesis death occurred about seventeen or eighteen days after the intercostal paralysis began.

From what has been said it will be seen that the changes in the spinal cord in my case agree very closely with those described by Dejerine and Dr. Abercrombie. The hæmorrhages, cell infiltration, and affection of the neuroglia which Dejerine observed were not present in my case. Dr. Abercrombie's description tallies with mine in this respect. The only changes I found in the grey matter were purely parenchymatous, no interstitial affection could be seen. Dr. Abercrombie's experience coincides with mine in this point too, and as his experience is based on an examination of seven cases I need hardly say that it carries much more weight than mine.

Through the kindness of Dr. Abercrombie I have had the opportunity of comparing his specimens with my own, I am consequently enabled to state that they agree very closely in almost every particular, though the cell degeneration in my case seems rather more advanced; this may

be due to the fact that Dr. Abercrombie's cases proved more rapidly fatal than mine.

Now, if we enumerate the cases of diphtheritic paralysis which have been published in which changes in the motor cells of the spinal cord are described, we find that there are fifteen in all.[1] This includes five cases published by Dejerine, two by Vulpian, seven by Dr. Abercrombie, and lastly my own case.

Against these we have to put the negative results obtained by Hermann Weber, Oertel, Pierret, and Vulpian, making four in all.

We must omit from our calculation cases in which no mention is made of a microscopic examination of the spinal cord. Moreover, those cases where the palate was the only part paralysed, are open to the objection that the paralysis may have been due to local causes only. At any rate this possibility cannot be excluded.

These objections apply to the cases of Charcot and Vulpian, Lorain and Lépine, Liouville and Leyden. How, then, are we to account for the negative results obtained by Hermann Weber, Oertel, Pierret and Vulpian? I confess that this is very difficult in the case of such excellent observers. But I would suggest that in proportion to the length of time the paralysis has existed before death we may expect to find greater or less changes in the motor cells.

In rapidly fatal cases of paralysis the cell changes may be possibly so slight as to escape detection even by competent investigators. It was long before the spinal lesion of progressive muscular atrophy and infantile paralysis obtained full credence, but I believe that, like these diseases, diphtheritic muscular paralysis will be shown to depend upon a "poliomyelitis anterior."

[1] Since this paper was written similar changes have been found in a case of diphtheritic paralysis by Dr. Mott, of Liverpool.

EXPLANATION OF PLATE IV.

Plate. IV.

Fig. 1

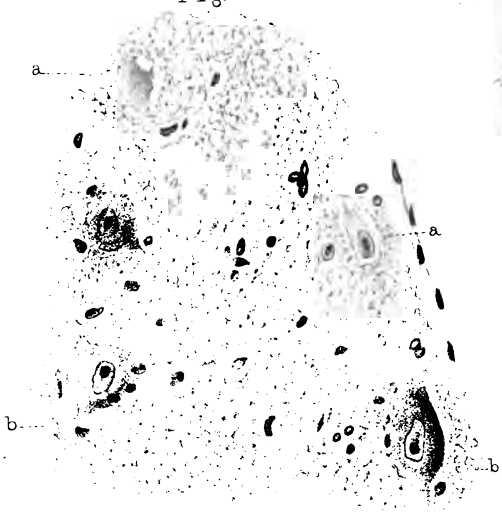


Fig. 2

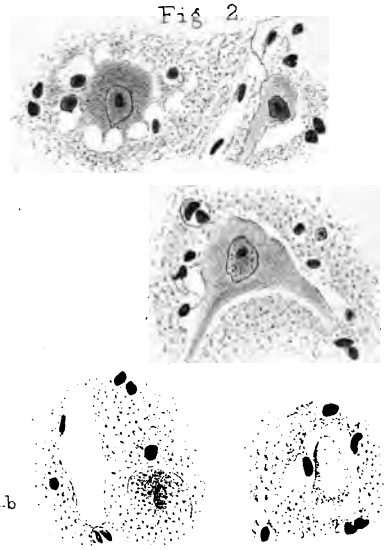
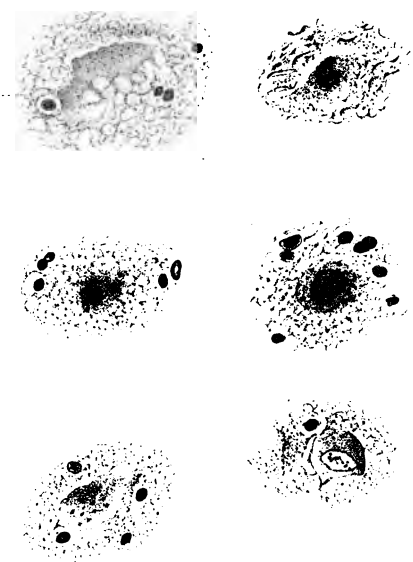


Fig. 3.

Fig. 4.



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DESCRIPTION OF PLATE IV.

(Pathology of Diphtheritic Paralysis. PERCY KIDD, M.D.)

FIG. 1.—Part of anterior horn, mid-dorsal region, showing contrast between indistinct shrunken nerve-cells (*a a*) and healthy ones (*b b*). × 300.

FIG. 2.—Individual nerve-cells from anterior cornua, mid-dorsal region, in different conditions of degeneration; some granular, globular, and nucleated; others pale, indistinct, and showing no nucleus. (*x*) One large healthy cell from same cornu. × 300.

FIG. 3.—Degenerate nerve-cells of different kinds, all of small size. One large healthy cell. Dorsal region. × 300.

FIG. 4.—Degenerate nerve-cells from lower dorsal and cervical region. One pale irregular nerve-cell shows vacuoles in its protoplasm (*v*). × 300.

Fig. 1

Fig. 2.

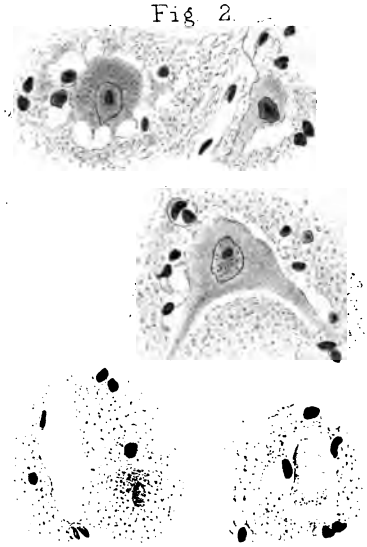


Fig. 3.

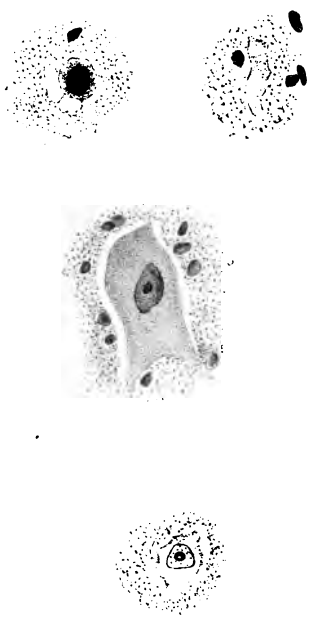
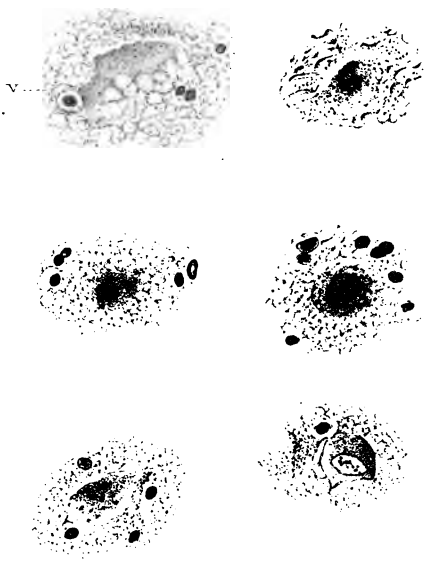


Fig. 4.



A SECOND CASE

OF

MALFORMATION OF THE LEFT SHOULDER-GIRDLE;

REMOVAL OF THE ABNORMAL PORTION OF BONE.

WITH

REMARKS ON THE PROBABLE NATURE OF THE DEFORMITY.

BY

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(Received February 12th—Read March 13th, 1883.)

In the sixty-third volume of the 'Transactions' is a communication which we had the honour of laying before the Fellows of this Society. It is "an account of a Specimen of Congenital Malformation of the Bony Thorax, Spinal Column, and Left Scapular Arch removed from the body of a woman, thirty-two years old, with remarks on the probable nature of the deformities." The malformation of the shoulder girdle, it may be remembered, con-

sisted of a triangular bridge of bone stretching between the spinal column and the scapula. Since then a child, eight years old, with an apparently similar, if not identical, malformation of the left scapular arch has come under the care of one of us (Mr. Willett) at St. Bartholomew's Hospital. The recurrence of a deformity which so recently appeared to be unique, the additional light which we believe it throws upon the nature of the malformation, and the fact that it admitted of successful performance of a surgical operation are our grounds for again bringing the subject under the notice of the Society.

The patient, Violet F., was admitted into Sitwell Ward, July, 1880, for the growing out of the left shoulder blade and lateral curvature of the spine. The mother died of consumption soon after the birth of the child, and the history obtained from the father was unsatisfactory and incomplete. He stated that the child had a nævus at birth; that when two years old she fell and dislocated her left shoulder, and that he, being then in the Army Service Corps, attended to the dislocation himself. Two years later the child hurt the same shoulder when at school. Since that time the deformity of the shoulder has been noticed and is said to be getting worse.

The child (see Plate V) who measures three feet seven inches in height, stands with her body tilted over to the left side, so that the plummet line from the occipital protuberance crosses the posterior superior spine of the ilium, and passes a little internal to the middle line of the left leg. The lumbar anterior curve is nearly normal; the dorsal posterior curve is much diminished, especially at its upper part where it appears almost to be absent. The lower half of the dorsal spine inclines to the right side, the upper half to the left side. The cervical spine inclines to the right. The left scapula is raised one inch higher than the right. Its upper angle, concealed beneath the trapezius, is three inches from the occipital protuberance; its lower angle is opposite the seventh rib which is raised nearly one inch higher than the right.

Its movements are not as free as those of the right scapula. Such movement as exists takes place at a hinge, which is situated at the junction of the scapula and the abnormal bridge of bone. This process of bone is of a triangular shape with its base attached to the scapula and its apex to the spinal column. It measures about one inch and a half in length and the same in breadth at its base. It tilts the posterior border of the scapula backwards and outwards, so that a prominent ridge is produced where the scapula and process of bone meet. It is at this ridge that the hinge-like movement occurs. The left trapezius is tense and stands out prominently, and immediately beneath its anterior margin is felt the superior border of the scapula. The inner curve of the left clavicle has disappeared, the external curve is prominent. The left arm when raised above the head does not come in contact with the side of the head; the right arm does. The right scapula is normal both as regards position and movement. Its upper angle is four inches in a direct line from the occipital protuberance. Its lower angle is on a level with the seventh rib. The ribs are apparently normal in number on both sides; the intercostal spaces are not increased in width. The shoulder-joints seem normal in all respects.

The operation.—On October 6th, 1880, chloroform having been administered, Mr. Willett made an incision three inches long parallel to the posterior border of the scapula, and another at right angles to it reaching to the spine. The abnormal piece of bone being thus exposed it was separated with the bone-scissors from the scapula, and having been lifted up was severed by bone-forceps from its connections to the spinous processes of the seventh cervical and first dorsal vertebræ. The operation, which was attended with but little hæmorrhage, was performed antiseptically. A drainage-tube having been inserted the edges of the wound were brought together with silver sutures. The patient progressed favorably. On the 13th the drainage-tube was removed and the

wound appeared almost healed. On the 30th passive movements were begun and the patient was discharged in good health, with some improvement of the shoulder, and with a mechanical support to keep the shoulder in place.

The portion of bone removed at the operation (see Plate VI, fig. 1), has the form of an irregular triangle with a truncated apex by which it had an osseous attachment to the seventh cervical and first dorsal vertebræ. At its base is a layer of cartilage half an inch in thickness by which it was attached to the scapula; the thin lamella of bone seen along the outer border of the cartilage being a part of the scapula itself, which in this situation projected slightly backwards, as it were, to meet the bridge of bone (a wedge-shaped piece of bone has been cut out from the base for the purpose of microscopical examination). The bone measures, excluding the layer of cartilage, $1\frac{3}{8}$ inch in length. Its breadth at its base is $1\frac{1}{4}$ inch; at its truncated apex $\frac{3}{4}$ of an inch. It varies from a $\frac{1}{4}$ to $\frac{3}{8}$ of an inch in thickness. The layer of cartilage connecting the bone to the scapula forms a gentle curve with its convexity backwards. It was this curve that gave rise to the prominent ridge that existed, as related in the history of the case, at the apparent junction of the bone with the scapula. The layer of cartilage is slightly thinner at the most prominent part of the curve than where it was connected with the bone or with the scapula. This thinning takes place at the expense of the cartilage on the concave side, so that the appearance of a slight vertical sulcus is produced in this situation. The sulcus is best seen on horizontal section (see Plate VI, fig. 2). It was at this spot that the hinge-like movement before alluded to evidently took place.

The bone, without taking the cartilaginous portion of the specimen into consideration, is slightly concave from base to apex, and nearly flat from above downwards. Its anterior or deep surface, except near the apex where it forms two prominent ridges, an upper and a lower with a deep groove intervening, is flat. The upper and lower

borders of the bone are rounded, the upper border being slightly expanded where it joined the spine of the seventh cervical vertebra. The bone is covered with periosteum. Muscular fibres are still seen attached to its anterior surface along the line of its junction with the cartilage, and along the outer third of its upper and lower borders. Muscular fibres are also seen attached to the concave surface of the cartilage. These fibres are the remains of the muscles cut through at the operation for the removal of the bone, probably the serratus magnus, levator anguli scapulæ and the rhomboids. On section (see Plate VI, fig. 2) the bone is seen to be composed of cancellous tissue a quarter of an inch in thickness enclosed in a thin outer layer of compact bone. Microscopic examination of a horizontal section carried through the bridge of bone, the intervening cartilage and the portion of scapula removed at the same time shews that ossification was advancing towards the middle of the cartilage from both the bone and the scapula.

Remarks.—That the abnormality of the shoulder girdle in this patient was similar to that in the specimen shown at this Society in 1880 (see 'Transactions' vol. lxiii Plate v), there can, we think, be little doubt. On comparing the portion of bone removed at the operation with the bridge-like piece in the deformed thorax, the resemblance that exists between them is at once apparent. On examination of the second patient before the bone was removed the state of the parts was so similar to that found in the first patient that a diagnosis of a like condition was at once made. At the operation, moreover, the connections of the bone to the spine and to the scapula, seemed, as far as could be made out, identical with those of the bridge-like piece belonging to the deformed thorax.

The occurrence of a similar deformity so soon after the first, which at the time appeared to be unique, is undoubtedly of much interest, especially as we think the slight difference observed in the second case throws additional light on the morphological significance of the abnormality. The difference to which we refer is in the attachment of

the bone to the scapula in the second case by cartilage. In the first case it will be remembered the attachment was by bone; although a sulcus containing cartilage in the recent state, made it probable that here also the union had at one time been entirely cartilaginous.

In our former communication we may remind the Society we suggested several theories, that seemed to us more or less probable, to account for the presence of the abnormal bridge of bone; and that we classified them according as they could be based upon one or other of the following hypotheses :—1. That the bone was formed in connection with the vertebræ, and afterwards became anchylosed to the scapula. 2. That it was originally a scapular element, and became secondarily fused to the spine. 3. That it was formed independently both of the scapula and the spine.

As the arguments for and against these theories were then discussed at some length we shall not further refer to them, but shall confine our attention to a consideration of the scapular theory which the present case seems to strengthen.

We are inclined then to regard the bridge of bone as an abnormal development of the supra-scapular epiphysis which normally exists as a narrow ridge of bone along the posterior border of the scapula, and consequently as homologous to the supra-scapular bone of some of the lower vertebrata. The points that we think favour such an origin are :—

1st. The apparent continuity in the first specimen of the bridge of bone with the supra-scapular epiphysis of which it appears to be an overgrowth. 2nd. Its cartilaginous attachment in the second specimen to the scapula. 3rd. The absence of analogy between it and any known form of exostosis; and the difficulty of explaining how if it were an exostosis from a vertebra it could have become secondarily attached to the scapula. 4th. The insertion of certain muscles into it showing that the abnormality occurred at a very early period of development. 5th. The

abnormal condition in the first specimen of the scapula itself; and the presence of other concomitant malformation of evidently congenital origin.

In our first specimen the bridge of bone is united by its base to the middle third of the posterior border of the scapula. On the posterior aspect this union is osseous, except for an inch above where there is a slight fissure filled in the recent state with soft tissues, and below where the small foramen is now seen. On the anterior aspect this union is cartilaginous, the situation of the cartilage before maceration being now indicated by a sulcus running vertically between the bone and the scapula.

Such a condition of the parts suggests an epiphyseal origin.

Now, along the lower third of the posterior border of the scapula the supra-scapular epiphysis is clearly united, the line of union being indicated by a distinct ridge. On tracing this ridge upwards it appears to be continued into the foramen before mentioned, and through it into the sulcus between the bridge of bone and the scapula; demonstrating, if this be so, that the sulcus itself is a continuation of the epiphyseal line, and that consequently the bridge of bone, which is, moreover, continuous by its lower angle with the united portion of the epiphysis, either is a part of the epiphysis itself greatly over-developed, or if formed from some other source has at least now become intimately blended with the epiphysis. The accompanying drawing (see Plate VI, fig. 3) which represents the condition of the parts is intended to shew these relations. Had the bridge of bone merely become united to the epiphysis some indication of such a union having occurred ought surely to exist; but from the epiphyseal line (the sulcus between the bone and the scapula) to the spinal column the bone appears of one uniform piece and were it not for its spinal attachment, its epiphyseal origin we think could hardly be called in question. We shall refer again to this point, however, later on. Now, that an overgrowth of the supra-scapular epiphysis may occur, is proved, we think,

by specimens in the Museums of St. Bartholomew's Hospital and of the Royal College of Surgeons. Series A 138 St. Bartholomew's Hospital Museum, is the left scapula from an anencephalous fœtus. An overgrowth of the epiphysis has occurred at the lower third of the posterior border. The opposite scapula presented a like deformity. No. 3287, Royal College of Surgeons' Museum, is a right adult scapula in which a well-marked outgrowth, evidently epiphyseal, has occurred at that part of the posterior border that corresponds to the triangular surface at the base of the scapular spine. Scapulæ showing similar deformities but in a much less degree are not so very uncommon. One is contained in the Museum of St. Bartholomew's. In No. 13 normal osteological series, Royal College of Surgeons' Museum (a right scapula and humerus), a well marked outgrowth of the lower third of the supra-scapular epiphysis has occurred and ossification in it has begun. The rest of the epiphysis is normal and not ossified. In No. 65, Teratological series, Royal College of Surgeons' Museum (the skeleton of a fœtal kitten) the supra-scapular epiphysis on both sides is apparently prolonged backwards, and is attached to the spine of the second dorsal vertebra. The parts are somewhat shrunken from the action of the spirit. There is no mention of the abnormality of the epiphyses in the account of the specimen in the Catalogue. No. 229 Teratological series, Royal College of Surgeons' Museum is a similar specimen to the preceding.

In the case now before the Society the base of the bridge of bone was united to the middle third of the posterior border of the scapula (which projected backwards, as it were, to meet it) entirely by cartilage. This cartilage consists of a smooth, uniform layer of the same breadth as the bridge of bone on the one hand and the scapula on the other; it is covered on its anterior and posterior aspect by a layer of perichondrium continuous with the periosteum covering the scapula and the bridge of bone; and on section ossification is seen to be advancing towards the centre of the cartilage, from both the bridge

of bone and the scapula. There is no trace, therefore, in this cartilage of any union having taken place between the cartilaginous border of the scapula and the cartilage of the bone; in other words there is no evidence that the scapula and the bridge of bone have ever been separate. Hence the cartilage in which the bridge of bone was developed would appear to have originally formed part of the general cartilaginous matrix of the scapula. But we know that the osseous material of the bridge was deposited as a centre distinct from that of the body of the scapula since a cartilaginous interval actually exists between them. We have here, therefore, as in the former specimen, all the appearances of the bone being an overgrowth of the epiphysis, but in an earlier stage of development. The osseous union in the elder patient and the cartilaginous union in the younger thus becomes intelligible; for the supra-scapular epiphysis normally unites with the body of the scapula about the twenty-fifth year of life; and as the one patient had reached her thirty-second year and the other had only completed her eighth, we should expect the bridge, were it the epiphysis, to have become united by bone in the former case, and to have remained united only by cartilage in the latter.

If the objection be here raised that the centre of ossification for the supra-scapular epiphysis does not normally appear until about the sixteenth to the eighteenth year, it may be answered that we are not dealing with a case of normal development; and we do not think it at all surprising, seeing the abnormal growth of the cartilage that has taken place, and considering the increased activity of the developmental processes which must have been going on, that ossification should have occurred at an earlier date than usual. We have seen, moreover, that similar instances of early ossification have occurred in the specimens of over-developed epiphysis—one in fœtal life—already alluded to.

Turning now to the vertebral end of the bridge of bone, its firm osseous fusion in both patients to the spines of the

vertebræ might seem to point to the bone being an exostosis of the spine, which had become secondarily united to the scapula, rather than to its being an abnormal supra-scapular epiphysis. It differs from an exostosis, however, in that it is not covered with cartilage except at the part where it is united to the scapula, in its flattened condition, in its evident growth in only one direction, in its broad base of attachment to the scapula; and in the insertion of muscles into it. Moreover, as far as we know, no case has hitherto been observed of an exostosis springing from one bone and forming an attachment to another bone, by or through its cartilaginous extremity; nor are we acquainted with any physiological or pathological process by which such a union could have been brought about. Lastly, regarded as an exostosis, it furnishes no clue to the concomitant malformation of the scapula and other bones which existed in the first specimen.

Setting aside then the idea of a spinal exostosis, let us consider how, on the assumption of its being an abnormal supra-scapular epiphysis, its intimate fusion to the spine can be explained. Now, the epiphysis that normally exists along the posterior border of the scapula in man presents a much higher grade of development in some of the lower animals, and in some even remains separate throughout life as a distinct bone—the supra-scapula. Figs. 4, 5, and 6 in Plate VI represent some of the appearances presented by these parts in different animals. Fig. 4 shows the distinct supra-scapular bone in the shoulder girdle of the frogs and toads, in some species of which it projects so far as to meet its fellow of the opposite side across the middle line of the spine. It is true that the supra-scapular bone is not, as a rule, fused to the spine, but instances of such a union are not wanting, as for example, in the placoid fishes. (See Plate VI, figs. 5, 6.) In fig. 5 a front view of the scapula, supra-scapula, and one of the vertebræ of the skate is represented; fig. 6 is the side view of these parts. Both are copied from Professor Parker's work on the shoulder girdle.

As the supra-scapular bone then, is admitted by all to be the homologue of the supra-scapular epiphysis of man it, of course, follows that the bridge of bone if it is, as we have tried to show, an abnormal development of the epiphysis must also be the homologue of the supra-scapula of animals. But we have seen that the supra-scapula of animals presents all grades of development, from a mere rudiment to a distinct bone united to the spine as in the skate. We have further seen that the supra-scapular epiphysis in man may actually present an abnormal backward development comparable to that, say, of the rabbit. It is advancing, therefore, but one step further to compare the bridge of bone in our specimen to the distinct supra-scapula of the frog, and but one step further still to conceive it united to the spine as in the skate.

If our scapular theory, then, be correct we are no longer met with the difficulty of having to explain how one bone can have become fused to another; for we must regard the bony bridge not as a mere outgrowth or growing backwards of the epiphysis, but as having existed in its present form, though of course in an unossified condition, from the time of the differentiation of the cartilaginous shoulder girdle from the mesoblast; and therefore as having had an attachment to the spine from the earliest period of its development. When we remember how similar in all early vertebrate embryos is the primitive cartilaginous rod from which the shoulder girdle is developed it would not seem that so very grave a departure from the normal developmental process were required to produce the malformation. We have only to conceive a slight extra-differentiation of the mesoblast in a backward direction to have the primitive cartilaginous rod reaching to the spine. Analogous processes, moreover, are not wanting in the human body, as, for instance, in the not infrequent presence of a cervical rib (as in birds) which as is well known is due to the over development of the anterior part of the transverse process of the seventh cervical vertebra—the homologue of the thoracic ribs; the

extra differentiation of the mesoblast extending in this instance in a forward direction so that in extreme cases the rib may reach the sternum.

If, therefore, by a forward extension the anterior part of the transverse process—the homologue of a rib—may be developed into an actual rib, as in birds, why may not the epiphysis—the homologue of the supra-scapula—by a similar but backward extension be developed into an actual supra-scapular bone as in fish? If in one case, through extra differentiation, the transverse process of the vertebra may meet the sternum, why in another may not the epiphysis of the scapula through a similar process meet the spine? It is curious that in our first specimen a cervical rib should also have existed.

Given then a cartilaginous union between the scapula and the spine, there is no difficulty in explaining how in the course of development the ossific centres in the spinous process and the supra-scapula, *i.e.* the bridge of bone, would meet and become ultimately fused, leaving no trace of the former separation any more than in the analogous process of the union of the epiphysis and diaphysis in the development of an ordinary long bone.

The probabilities of the malformation having existed from a very early period are strengthened, we think, by the following facts:—

1st.—The attachment of certain muscles to the bridge of bone. Had it been simply an outgrowth from the spine or from the scapula, *i.e.* an exostosis, we should have expected to have found the muscles merely displaced by the growth, certainly not inserted into it as they were in both specimens.

2nd.—The rudimentary condition in the first specimen of the scapula itself a fact clearly shewing, theory aside, that some developmental defect has *actually* occurred in the formation of the shoulder girdle.

3rd.—The concomitant malformations of the spine, ribs and clavicle in the first specimen, which it may be remembered were shewn in our former communication to have

occurred at a very early period in the development of the embryo.

In conclusion if we admit that the bone is a supra-scapula, it would appear that in both of these cases we are dealing with a reversion of the human shoulder girdle to that of a lower type. We confess, however, that we have to seek far down in the scale of animal life before we find its exact homologue; and it may seem improbable that missing so many higher and intermediate forms it should revert to so low a type as that of the skate. These difficulties we think, however, are not greater than those which attend the theory of reversions in general, which theory nevertheless is admitted by most biologists to be the fittest solution for the many similar abnormalities of daily occurrence in the human body.

Note taken 21st February, 1883, of present condition of the patient.

Front aspect.—Head carried downwards, forwards, and to the left in an oblique direction, the point of the chin being 1½ inches to the left of the mesial line, and the left ear 1¼ inches above the level of the right. The left shoulder is also rotated slightly upwards and forwards.

Behind.—Scar of operation wound is very sensitive, but otherwise quite natural. Underlying the scar is a dense rounded band of tissue, of the feeling and consistence of muscle, stretching between the upper third of the posterior border of the scapula and the truncated stump of the spinous process, which remains distinctly prominent.

The cervico-dorsal spine is curved to the left, the lower half of the dorsal vertebræ to the right; with corresponding rotatory changes.

Movement of the scapula.—This bone rotates with considerable freedom; however, when the arm is manipulated the upward movement of the arm is checked when it reaches an angle of 145° with the trunk, apparently being held by the band of tissue above mentioned.

Those in charge of the child report that she uses the left arm as freely as the right in all respects.

In conclusion it would appear that greatly improved use of the arm has resulted from the operation, but that no appreciable change in the curvature of the spine can be noted.

DESCRIPTION OF PLATES V AND VI.

Case of Malformation of the Left Shoulder Girdle. (Mr. A. WILLETT and Mr. W. J. WALSHAM.)

PLATE V.

The child from whom the piece of bone was removed by Mr. A. Willett. (From a photograph.) Back and front views, before the operation.

PLATE VI.

FIG. 1.—The bridge of bone removed at the operation, front view.

FIG. 2.—Ditto, horizontal section.

FIG. 3.—Condition of parts in 1st specimen (see vol. lxiii, Plate v).

A. The bridge of bone, continuous with :—

B. The united supra-scapular epiphysis.

C. The body of the scapula.

FIG. 4.—Scapula and supra-scapula of frog. St. Bartholomew's Hospital Museum, S. 28, No. 248.

A. Scapula.

B. Supra-scapula.

FIG. 5.—Scapula, supra-scapula, and vertebra of the skate (from Professor Parker's 'Monograph on the Shoulder Girdle'), front view.

A. The vertebra.

B. The supra-scapula.

C. The scapula.

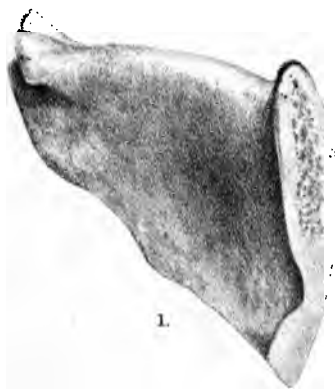
FIG. 6.—Ditto, ditto, side view.

Fig. 1.

Fig. 2.

R. Mintern, lith.

Mintern Bros. imp.



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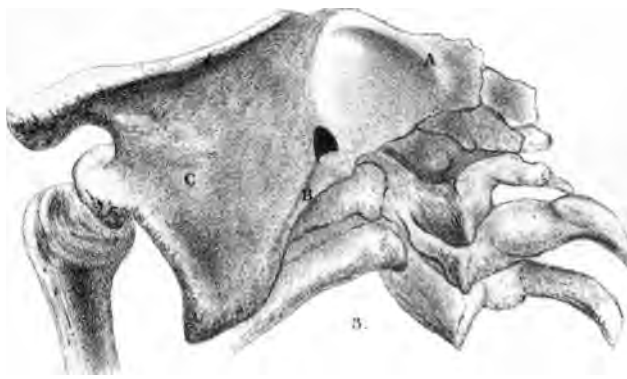

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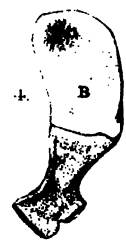

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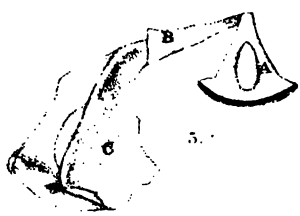

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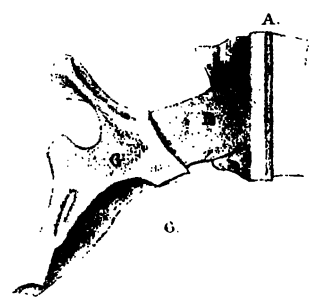

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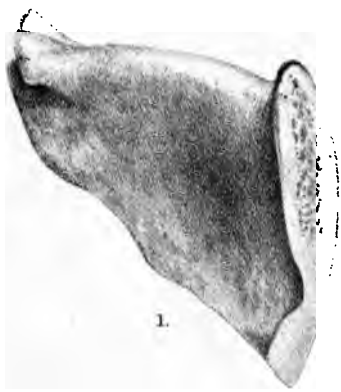
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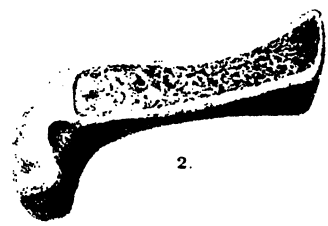
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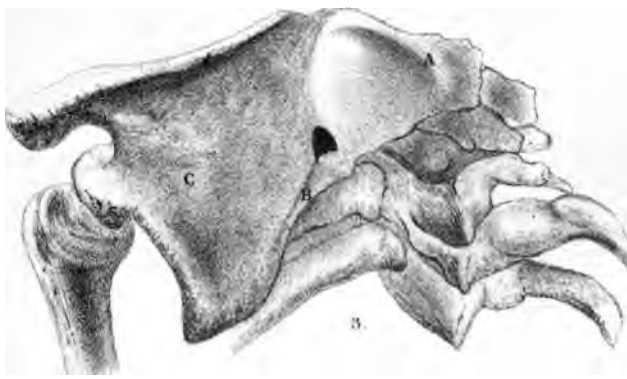
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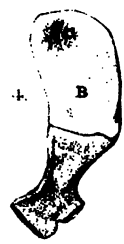
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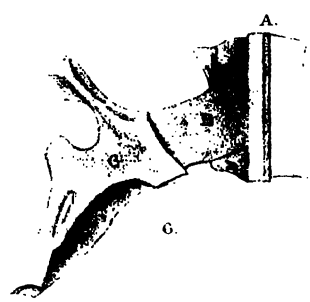
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ON

CASES DESCRIBED AS "ACUTE RICKETS"

WHICH ARE PROBABLY

A COMBINATION OF SCURVY AND RICKETS,

THE SCURVY BEING AN ESSENTIAL, AND THE RICKETS A VARIABLE, ELEMENT.

BY

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(Received March 13th—Read March 27th, 1883.)

THIS paper is a contribution to the study of a disease occurring in young children, of which several cases have been recorded in recent German and English literature, but of which hitherto, with one exception, no satisfactory account of the morbid anatomy has been given.

I propose (1) to narrate a typical case, (2) to give an analysis of principal symptoms in the recorded cases and in cases which have come under my own observation, (3) to give the results of post-mortem examinations, and (4) to discuss the etiology and affinities of the disease.

(1) *Typical case.*—A boy, aged fifteen months, was seen in the month of December when the following note

describes his condition :—The child has an excessively pale, sallow complexion and is flabby, although he has a moderate covering of fat. There is no sign of nervous disease nor of visceral disease except that the liver is perhaps larger than normal, extending to two fingers' breadth below the thoracic margin as the body lies in the horizontal posture. The bowels have acted once or twice daily; a stool which has been saved is greyish-brown in colour and a little slimy. There has been no vomiting; the tongue is clear. He has cut his two lower incisors; the gums are natural with the exception of a minute erosion in the upper gum opposite the cutting edge of one of the lower teeth. The boy is continually moaning and when approached he screams and still more when he is touched. It is difficult to describe a cry, but it is sufficient to say that in this case it suggested deep-seated pain connected with bones and not brain disease. And it is clear that the dominant symptoms are related to the bones. The boy is rickety; there is some beading of ribs although the thorax is not grooved, his epiphyses are a little enlarged and he has only two teeth, but rickets is not sufficient alone to explain his condition. He lies on his back and scarcely moves the trunk though he frequently turns his head from side to side. Both radii are enlarged at the lower end but the right more so than the left, not only in circumference but in vertical measurement to a slight amount. It, in fact, suggests a rickety enlargement plus slight thickening extending upwards for perhaps an inch. The child cries wherever he is touched, but the mother has noticed his right wrist notably more tender than the other for a day or two. The skin is pale in the neighbourhood and there is no special heat to be felt. The upper limbs are not bent. The left thigh is kept half flexed. Both the left thigh and leg are slightly swollen so that the contour of the limb is different from natural, assuming in the thigh rather a cylindrical shape. It is of the same colour as the other limb and does not feel hotter than any other part of the body. There is no fluctuation and no sign of

effusion in the knee or ankle-joint. The child screams so much that one cannot examine thoroughly, but in spite of his thigh being flexed there does not seem reason to suspect hip-joint trouble.

The epiphyses at the knee and ankle are enlarged.

The right thigh is natural in the sense of there being no swelling. Besides the slight enlargement of epiphyses at the knee there is a tendency to knock-knee on the right side. There is no general swelling of the right leg, but there is a little thickening to be appreciated down the shaft of the right tibia.

There is profuse sweating about the head. There is nothing special about the head itself, except that there is a suspicion of slight thickening of the frontal in front of the fontanelle.

The boy's temperature taken in the rectum is 101° F., 7 p.m.

The history was as follows:—He was the only child of his parents, a young couple in good circumstances, and living in a healthy dwelling. The father is in good health, but the mother is a thin, poorly developed woman, without, however, any history of particular ailment. The child had been born before term and had in its babyhood occasional stuffiness of the nose, but beyond this there was nothing to suggest congenital syphilis, though it is of course difficult to negative its possibility. During the first six weeks he was said to be a vigorous child. For that period he had his mother's milk, then it entirely failed, and from that time until when I saw him he had been quite deprived of fresh food. At first his diet consisted of Robinson's grits and Swiss milk, then of baked flour, then of Nestlé's food, then of Robb's biscuits, then of Liebig's extract, and finally of Swiss milk and saccharated lime water. He had been considered a fairly healthy child. He had not suffered from cough; his bowels had acted not more than twice daily, although the nurse admitted that the evacuations were often unduly offensive; he had had scarcely any vomiting. He had cut his first

tooth at twelve months and his second at thirteen months, and these were all he had. His mother admitted that he had been always a pale child, and that although his limbs had been fat they had been thick near the joints, also that he had had much head sweating since he was three months old.

The child had been able to sit up well and stand with assistance at thirteen months old. Five weeks ago he ceased to do either, and then it was noticed that the left leg was swollen especially about the ankle. At this time also he became very peevish and would shriek if he were touched, and often even if he were approached. The boy was then taken to a well-known bone-setter, who informed the parents that one of the bones of the spine was out, and that an operation was needed to restore it to its proper position. Five days later the said operation was performed, but as the swelling of the left lower limb increased, and the right wrist became swollen and the right hand dropped, and no explanation was forthcoming except the vague suggestion of possible paralysis, a further opinion was desired, and the child's condition was found as above described.

As to diagnosis, it was obvious that the child was the subject of rickets to a moderate degree, and equally obvious that rickets alone was insufficient to explain all his symptoms.

On the ground of certain post mortems which are subsequently detailed, the opinion was formed that in this case there was under the periosteum of the left femur and tibia an effusion of blood, and that the extreme tenseness of the limb was due to blood extravasation in the deeper muscular layers with the serum filtered out into the more superficial parts.

It seemed likely that there was also some slight blood effusion immediately around the shaft of the right tibia and also in the neighbourhood of the junction of shaft and lower epiphyses of the right radius. The view held was that the boy was suffering from the supervention of

scurvy on rickets, albeit there was no trace of sponginess of gums. The treatment suggested was to surround the whole of the left lower limb and the right leg with wet compresses which had been thoroughly wrung out. These were to be surrounded with dry cloths rather closely applied, and the compresses were to be changed every two hours.

A complete change was made in the diet. He was ordered daily the juice of a quarter pound of raw beef sweetened a little. He was also to take a pint and a half of cow's milk daily, to which was to be added in his alternate meals at one time a third part of strained gruel, and at another a third part of barley water, and finally two teaspoonfuls of orange juice were to be given daily. It was also ordered that for at least half an hour twice daily the boy's crib should be placed near the fire, and that the window of his room should be opened wide and the door likewise, so that he should have a free play of fresh air around him, precautions being taken about close fitting garments. The directions were most loyally carried out, and in three days there was a notable change in the child. The compresses had certainly been a comfort to the child, so that on the first night of their application he had been a great deal quieter than before. He had taken the fresh food most greedily, and it had agreed with him. His tongue was almost clean and a healthy evacuation had been passed. His rectal temperature had sunk to 99·4°. The left lower limb was less tense and less tender, and the right leg was better.

After this the improvement was progressive. It was several days before any of his urine could be saved, then it was found pale, clear, neutral, free from albumen or excess of phosphates. After about a fortnight the tenseness of the soft parts of the left lower limb had disappeared, and then it was easy to appreciate that the left femur was thicker than the right, and in less degree that the left tibia was thicker than the right. The thickening gradually diminished, but even at the end of six weeks a slight difference could just be felt between the two femora.

Before this time the swelling of the lower end of the right radius had also considerably subsided, so that in fact there was no difference between the two wrists. The compresses were omitted at the end of a fortnight from the date when he was first seen, and he began then to make efforts to raise himself and to move his limbs about. A little gentle shampooing of the lower limbs and back with oil, and likewise douches of first tepid and then cold sea water were commenced at the end of a month. No physic was given to him except at first a single, then two teaspoonfuls of cod-liver oil daily, and an occasional powder of rhubarb and soda. Within eight weeks from the date when first seen, the boy, whenever allowed, would get upon his knees and could stand with a little support; he was of a ruddy colour, and his skin and muscles had become quite firm. He was still not allowed to bear the weight of his body on account of the slight knock-knee on the right side, but he was carried out every day.

(2) *Analyses of principal symptoms.*

The material from which this analysis is made consists of thirty-one cases. Of these nineteen have been already published. The majority of these cases have been recorded in German medical literature under the title of "Acute Rickets" (Möller, Bohn, Forster, Hirschsprung, Senator, Petrone, Fürst). Some account of the disease is also given by Steiner and Baginsky in their respective treatises. Individual cases are referred to by these authors, but the accounts of them are too brief to admit of incorporation in the accompanying table. Stiebel's description of acute rickets, which differs somewhat from the clinical picture given in common by the other authors, is also not available for this analysis, because of the absence of cases reported in detail. One case, briefly recorded under the title of infantile scurvy, by Dr. Ingerslev, also belongs to our group, and is, indeed, quoted by Hirschsprung *à propos* of his own case.

Sir William Jenner in his lectures refers to the occasional abrupt or acute onset of rickets, but does not specifically describe any case like those in question. The first English case on record is in the 'Pathological Transactions' for 1876. It was under the care of Mr. Thomas Smith at the Hospital for Sick Children, and is described under the provisional title of hæmorrhagic periostitis.

Dr. Gee, in the 'St. Bartholomew's Hospital Reports' for 1881, has given brief notes of five cases, which he classes under the head of *osteal or periosteal cachexia*, pointing out the existence of an obscure bone lesion and of a marasmic condition not explicable by rickets or congenital syphilis. It will be found that these cases come within the group now under consideration.

In the 'Lancet' for November, 1878, and again in July, 1882, Dr. W. B. Cheadle has described two cases of the same kind which came under his care, and has referred to others. Dr. Cheadle has given reasons absolutely conclusive, as far as his own examples are concerned (in which spongy gums were a marked feature), for the doctrine that the disease in question is a combination of rickets and scurvy; and his papers are in every way the most important contribution to the clinical side of the subject.

A French writer (Montfalcon) in the article on "Rickets" in the 'Dict. des Sciences Médicales' (1820), gives a short paragraph on the complication of scurvy with rickets. He narrates a case of a girl, aged ten years, who was certainly the subject of scurvy of the gums and skin ecchymoses, and who had some obscure joint (?) or bone (?) affections. It seems extremely probable that the latter were scorbutic also, and the case is of some value in support of the view subsequently maintained in the following paper, that the essential features of acute rickets are truly scorbutic; but it is to be noted that the writer of the article in question gives no reason for the view that his patient was the subject of rickets, and, indeed, throughout his article uses the term rickets in an extremely vague and indeterminate sense.

Further, this patient was much older than those (chiefly infants) the reports of which have already been referred to, and for this reason is not suitable for the analysis.

Eleven cases have come under my own care, and one has been communicated to me by Mr. Shoppee, and these with the nineteen before published constitute the material from which the analysis is made.

The invasion of the disease may be gradual or abrupt. After a few days inexplicable fretfulness, or several weeks' manifestations of pain, without obvious cause, when the limbs are touched, the child is rather suddenly taken off its legs, with more or less swelling of the lower limbs. In twenty-two cases out of thirty-one the thighs have been attacked, and in the same number (many of them of course the same cases) the legs. In two other cases the lower limbs are spoken of as affected without definite description. In a typical case both lower limbs, though to an unequal degree, are swollen, tense, and shining, the skin is generally pale, but may be reddish in colour, not generally hotter to the feel than the rest of the body, sometimes indeed colder. There is a varying amount of œdema, often more over the thigh than the leg or the foot. The tenderness on pressure is extreme; the child cries not only on movement but even on the approach of the nurse. In a severe case there is often continuous moaning as though the child were in constant pain. The child lies sometimes with one or both limbs flexed, but in the more severe cases with the limbs extended, or extended and everted in an immobile condition, which has been called pseudo-paralysis. In less severe cases, even at the outset, and in more severe cases after the partial subsidence of the subcutaneous swelling, it is not difficult to appreciate that there is a cylindrical swelling which envelops the shaft of the femur to a varying degree. The longitudinal extent of this swelling may vary, it may be very slight and be confined to the neighbourhood of the junction region between the shaft and epiphysis, or it may surround the whole of the shaft. In the most severe

cases of all, of which I have seen two examples (XIX and XXVI), soft crepitus was to be obtained just below the hip-joint. This no doubt is a late and very severe phenomenon, but from considerations afterwards to be referred to, I think it probable that it may occur not unfrequently, and owing to the excessive tenderness of the patient, which interferes with careful examination, may escape detection. In Dr. Fürst's case (XI), after the conclusion of the illness, a sharp angular bending with some thickening was found in both femora though the exact site is not indicated. Nevertheless, the ordinary sequence of events with respect to the thigh is, first swelling of the whole of that part of the limb, then subsidence of the affection of the soft parts, allowing the thickening around the bone to be appreciable, then very gradual subsidence of the bone thickening, leaving the limb without deformity. In one of Möller's cases (IV) and in Förster's case (VI), after the illness a marked increase of the length and growth of the limbs was noticed. This is, however, by no means a constant occurrence.

The affection of the legs was occasionally present without involvement of thighs, but whether it existed alone or in combination with the thigh affection, it was in all the cases in my experience less severe than the thigh affection. Here, also, it may be said, in general terms, that the thickening radiates from the junction areas of shaft and epiphysis. The fibula is always much less affected than the tibia. In one of Möller's cases (III) there was still a little thickening of the bones of the legs when the child died of atrophy, and in Hirschsprung's case there remained at the end of the illness a little "thickening of one ankle." With respect to the upper extremity in two of the cases already referred to (XIX and XXVI), there was soft crepitus obtainable below the knee. Probably also in another case (XXVIII) there had been some separation in this junction region, for at the end of the illness the shaft of the tibia was found displaced backwards to a slight extent just below the upper epiphysis. With

regard to the leg as with regard to the thigh, the rule is for ultimate resolution to take place without deformity.

Other bones may become affected either simultaneously or more commonly by subsequent involvement. In one of Dr. Gee's cases (XIII) there was some swelling over both scapulæ. The same condition was observed in one of mine (XXVI), the swelling being much more marked in the right than the left, and affecting especially the infra-spinous fossa.

The humerus was affected in nine cases, and the radius and ulna, either separately or conjointly, in twelve. In one case, viz. XIX, there was soft crepitus obtainable near the upper end of the humerus on both sides.

In general, it may be stated, with respect to the upper limbs, that the swelling of the soft parts was much less and the thickening along the shafts much more limited in extent. In the majority of cases, indeed, the swelling was limited to the epiphysial region; and it may reasonably be asked how was this to be distinguished clinically from ordinary rickety enlargement of the wrist for example? To this it may be replied, as in the type case, that probably some of the swelling is truly rickety but that there is something over and above the rickety swelling. (1) The appearance of the two wrists is often unlike; the enlargement above one epiphysis, being greater considerably than above the corresponding one on the opposite side; (2) the extreme tenderness is not present in simple rickets; (3) the pseudo-paralysis which often obtains in these cases does not occur in simple rickets; (4) the marked subsidence in a short time as in the type case is quite unlike the slow retrogression of an ordinary rickety enlargement.

With regard to the cranium, morbid conditions were found either during life or post mortem in eight cases out of the thirty-one. The exact amount which belonged to the illness in question is difficult in all cases to assign. Setting aside for the present the marked cranial bosses (Parrot's swellings) which were found in XXIV and XXIX,

and also the slight thickening near the sutures which obtained in XXVI, it seems probable that some slight swellings on the parietals in XXV, which were found post mortem to be due to sub-periosteal hæmorrhage, belonged strictly to the illness in question.

Further, in VII there appeared during the illness some swelling of both upper and lower jaws, and in XI some thickening of the zygomatic regions occurred, and likewise considerable tenderness of the occiput followed by some thickening.

Let us consider now the other structures involved in movement. With respect to the joints generally of the upper and lower limbs, although the phrase "painful joint affection" is more than once employed, there is nothing in any of the reported cases to lead us to believe that there was effusion into the synovial cavities. Senator, Fürst, and others, are most careful in their descriptions to exclude joint effusion. There was certainly no evidence of it in any of my cases.

The muscles generally in the severe cases were more or less wasted. It is difficult to estimate how much of this was due to antecedent rickets, but it is to be observed that several of the children had been in good condition before the illness began. In several the weakness of the back was most remarkable. No lesion of the bones was detected, but the prostration was often quite absolute. In Hirschsprung's case (VII) for a time there was great tenderness over the neck.

With respect to the skin and subcutaneous tissue, the anæmia in the severe cases was profound; besides the pallor there was noted sometimes a peculiar sallow, muddy tint in the complexion.

In one of Möller's cases (IV) sugillations appeared on one leg, and Hirschsprung says of his that "in several places the skin was tinged bluish red."

In one of Dr. Gee's (XIV) there was an appearance over the sternum like that produced by a "bruise."

In one of Dr. Cheadle's (XVII) there were unhealthy

sores on the wrist and finger, and it was stated that a blow on the thigh had left "a swelling for a considerable time."

In one of my cases (XXIII) extensive purpura appeared before the bone condition became manifest.

In one of Dr. Gee's (XIV) and in one of my cases there was ecchymosis into the eyelids, and in the latter case also there was for a few days slight proptosis of one eye. Some conjunctival hæmorrhage occurred in Mr. Shoppee's case (XXXI).

The tenseness of the lower limbs is in the very severe cases very considerable, but it is remarkable how completely as a rule the swelling passes away. In one of my cases (XXVIII) there was for a week or more a small, soft, fluctuating area just above the junction of the lower epiphysis of the femur with the shaft on the outer side of the thigh. This also underwent spontaneous absorption. In another case, which I believed to belong to the group, the affection was confined to one thigh. There appeared considerable swelling, which at one place on the outer side of the middle of the thigh gave at length some fluctuation. This was opened by my colleague Mr. Morgan, a little pus escaped, and the small diffuse abscess cavity very rapidly healed, leaving, however, considerable deep-seated thickening and (which could be appreciated after a time) marked thickening around the shaft of the femur. As this case is so exceptional in regard to suppuration, I have thought it wiser not to incorporate it with the others, the more so that as the other limbs were not involved there was no opportunity of comparing their progress to both typical cases, and so render its identity certain. It deserves further investigation whether the subcutaneous swelling may not occasionally in severe cases of the so-called acute rickets undergo a partial suppuration.

In almost every case very severe head sweating is referred to, but in several this symptom had existed from a very early period, and it may be questioned whether it

was special to the illness under consideration, although a very striking feature.

Here may be conveniently considered the subject of the body temperature in these cases, and some details must be given because in the reports there is some divergence of results. Senator in discussing acute rickets insists that pyrexia is a constant accompaniment, but this is certainly too sweeping a statement.

The case which best supports this view is that of Dr. Fürst (XI). Observations made every three or four days during a month, recorded some pyrexia lasting through that period. At the onset of the illness the evening temperature was 102·7°, and this was the highest recorded. Within the next five days 101·8° and 101·5° were registered, the first an evening the second a morning temperature. After this no temperature higher than 100·4° was recorded, and at the end of the month it was 99·8°. It is important to bear in mind that in Dr. Fürst's case during the first eleven days, there was progressive involvement of different limbs, viz. first the left thigh and leg and right leg, then the right forearm, then the right arm, then the left arm and forearm, and that even so late as the twenty-fourth day there was some infiltration and tension about the left arm. After the end of a month no further observations were taken as the child was improving. Six weeks after this a relapse occurred which affected both zygomatic regions and the right thigh—this thigh not having been previously attacked. The temperature rose to 102°, but in a fortnight had become normal again.

Petrone (X) gives the temperature of his case at the outset as 102·9°, but does not mention what it was subsequently. Hirschsprung (VII) states that in his case the illness commenced with febrile symptoms, and that there was marked but irregular pyrexia. In his case also there was successive involvement of left shin and foot, then of left forearm and head of humerus, then of right foot, then of upper, then of lower jaw, which afterwards relapsed.

In Mr. Thomas Smith's case (XIX) the temperature

was observed twice daily during the six days that the child was in hospital. The highest temperature was 101·4°. On another occasion it was 101·2° and at other times it ranged between 97·4° and 100·6°.

This case was very severe in regard to bone lesions, but I do not think there was proof of any fresh start of bone-mischief during the week that she was in hospital. Furthermore, the child was suffering from some bronchial catarrh, and post mortem a small patch of consolidation was found.

In the type case I have mentioned that the rectal temperature when I first saw the child was 101°. This was probably the twenty-sixth day of his illness. An intelligent nurse told me that there had been some fever for several days.

Three days later the rectal temperature was 99·4°, and subsequently taken every day was scarcely ever above 99°. There was no reason to suppose that any fresh bone-mischief supervened after the twenty-sixth day. In one of my cases (XXV), twenty-two days after the onset, the axillary temperature was 99·8°, and in another (XXVIII), ten days after the onset, rectal temperature was 99·6°. In several others, although the temperature was not taken, it was noted that the skin was cool. Bohn states that in his case (V) there was no fever, but the child was only brought to him one month after the onset.

In some of Möller's cases there was for a time a little local heat, but he states that there was no fever (I, II, III). In another case (IV) the temperature was not elevated at the onset, but at a later period the child was febrile, with some catarrhal conditions. In Dr. Cheadle's second case the temperature when first seen was 98°, and 99° is the highest degree recorded. In Dr. Cheadle's first case, during the six weeks that the child was in hospital the axillary temperature was either normal or subnormal, except on the day after admission, when it was 99·5°. When I saw this child in the out-patient room before his admission into the ward, his rectal tem-

perature was 103°. So far as the history could be relied on this was between two and three months after the onset of the illness. In both these cases of Dr. Cheadle's it is important to note that no new manifestation of bone affection appeared after they came under care, and a definite line of treatment was promptly adopted by him.

To sum up these somewhat divergent results, it is clear that Senator's statement that pyrexia is a constant accompaniment of the disease cannot be accepted. It is not considerable in amount, nor does it show any special hectic character, and for long periods it may be absent. A careful examination will, I think, leave little doubt that, setting aside some cases of intercurrent catarrhs, the pyrexia, when present, is related to the bone affection, and that, not so much in regard to extent as to the amount of tension present. Probably, with a fresh involvement of bone, if much stretching of periosteum occurs, there is a little fever, at all events for a short time.

Of the affections of the mucous membranes the most important is that of the gums. In fifteen out of the thirty-one cases there was some morbid condition present. In at least four of these there was sponginess with a tendency to bleed, and some putrid odour (IX, XVII, XXX, XXXI). Nine showed varying degrees of mouth affection (IV, V, VI, VII, XVIII, XX, XXI, XXIV, XXVII), from slight swelling confined to the neighbourhood of newly-cut teeth up to general swelling of both gums, and in one case of the lower lip also.

As to the date of appearance of the stomatitis in relation to the swelling of the limbs, in some cases it was antecedent to the limb affection (VII, XVII, XXVII, XXX) in others it occurred after the limb affection was well established (IV, XXI, XXXI), and in others it was probably simultaneous.

In one of Dr. Gee's cases (XIV), and in one of mine (XXV), the gums were not swollen, but there were small localised ecchymoses beneath the gum in the situations of the on-coming teeth.

It is very important to note that in six cases it is specially stated that there was no stomatitis (XI, XIX, XXII, XXVI, XXVIII, XXIX). It is almost absolutely certain that in a great many of the other cases stomatitis, or, at least, sponginess, was conspicuously absent; for the reports are given by authors who were fully acquainted with the occasional occurrence of this symptom in this group of cases (Senator, Möller, and others), and it is reasonable to believe that the frequent absence of sponginess of gums is the cause of the scorbutic hypothesis often being dismissed, or not entertained, or regarded as unimportant.

The details with respect to disturbances of the digestive tract are not very complete. In two cases (XXV, XXXII) the bone affection appeared to start after a severe attack of diarrhœa. On the other hand, in a great many of the cases the intestinal condition was considered healthy at the time of onset of the acute symptoms, and there was no notable disturbance throughout.

In the ninth week of the illness in one of Möller's cases (IV), severe diarrhœa with some bronchial catarrh occurred. In Hirschsprung's case (VII) there was for a time obstinate vomiting. This is very exceptional. In the majority of cases the appetite was maintained. In two of my cases (XX and XXI), children of four years and two years respectively, there was present the greatest antipathy to meat and vegetables, and one of Dr. Cheadle's patients (XVII), a boy aged thirteen months, had very great dislike to gravy and potatoes. But in these cases this dislike was long antecedent to the illness in question.

There is very little noted with respect to the liver. Slight enlargement of the spleen and lymphatic glands occurred in Petrone's case (X). The former I believe, is exceptional, for in several other cases, absence of enlargement of this viscus is specially noted. In one only of my cases was the spleen palpably enlarged (XXIV) in a child in whom there was some suspicion of congenital syphilis.

The spleen continued larger than normal after the bone affection had subsided, and possibly had existed previously.

The accounts of the urine are somewhat scanty. In one of Dr. Gee's cases (XVI) there was hæmaturia for a short time, and this was also noted in Mr. Shoppee's case. In Dr. Cheadle's two cases (XVII and XVIII) there was for a few days a definite trace of albumen. In one of Möller's cases (IV) and in Mr. Shoppee's (XXXI), for a time there was a considerable deposit of uric acid. Both Bohn and Hirschsprung specially note that in their respective cases the urine was normal, being free from albumen or excess of phosphates, and this also was the case in the type case given at the outset of this paper.

There is nothing important to note regarding the heart and lungs.

With respect to the nervous system, extreme fretfulness must be mentioned as a symptom so striking and constant that everybody who has recorded cases has dwelt upon it as something quite remarkable in this affection. In fact this is one of the five constituents, viz. pallor, wasting, immobility, swelling of limbs, and fretfulness, which, *with or without* swelling of gums, pretty nearly sum up the clinical whole of the so-called acute rickets. This special fretfulness is almost certainly related to the affection of the limbs.

The laryngismus stridulus and fits which occurred during the illness in Dr. Fürst's case (XI), and the tetany and laryngismus which occurred a short time before the onset of the bone symptoms in one of mine (XXVIII), belonged doubtless to the severe rickety diathesis which both these children presented. No manifestation of this kind occurred in the case of children who had been previously healthy.

There remains to be considered, in this section, the duration of the disease.

Slight differences of reckoning depend on whether the end of the illness is considered to correspond with the entire or partial disappearance of bone thickening, the child being well in other respects. Excluding the com-

paratively mild cases, there are the rather wide limits for the duration of one month and six months. The duration in the greatest number of cases is between two, and three and a half months.

It will be seen from the table that the disease is a very fatal one; no less than seven out of the thirty-one died. It is equally remarkable to note the slow but gradual, and apparently spontaneous, tendency to complete recovery which occurred in others, even when the cachexia had been very profound.

Two of the cases, viz. Bohn's (v) and Mr. Thos. Smith's (xix), appeared, from the history, to have passed through similar attacks many months previously.

(3) *Post-mortem appearances.*

Although the German physicians have given such careful accounts of the symptomatology of this disease under the designation of acute rickets, they have no description of the morbid anatomy to offer, and this accounts for their very vague and unsatisfactory explanation of its pathology.

Professor Möller in his second paper describes two cases. One of these (No. IV in the accompanying table) is fairly typical. The other was a marasmic child aged 15 months, who was brought with exophthalmos and sanious discharge from the nostrils, and who died from exhaustion. On post-mortem examination an extensive hæmatoma was found under the frontal and anterior parts of the parietals, extending down to the orbital plates of the frontal and to the ethmoid and into the orbits. The blood was believed to be derived from the inner surface of the bones of the skull in which no thickening was found, but the vascular canals of the inner surface of the frontal were considered to be wide and the bone itself somewhat porous. *No affection of the limb bones either during life or post-mortem was found.* The case can therefore in no sense be regarded as agreeing with the typical examples of so-called acute

rickets, of which the foregoing analysis has been given, albeit I am not prepared to say that it has no alliance with them.

It must in justice be stated that Förster made the shrewd guess with regard to the affection of the limbs in typical cases, that the essential condition was a sub-periosteal hæmorrhage; and Möller, in some parts of his paper, seems to have come very near to that view, but in his summing up he speaks in a vague way of "acute rickets as being solely a developmental disease in which there is a precipitate and tumultuous bone growth, which at times destroys the organism, killing through exhaustion, but most often is overcome leaving behind it a marked increase in the length of the limbs."

A much simpler doctrine will I believe be elicited from the study of the clinical phenomena in the light of post-mortem examination.

I have made three autopsies on cases of the disease under consideration. The first was on a child under the care of Mr. Thos. Smith at the Hospital for Sick Children in February, 1875. Mr. Smith has recorded the case in the 'Pathological Transactions,' vol. xxvii, p. 219, under the title of "Hæmorrhagic Periostitis of the Shafts of several of the Long Bones with Separation of the Epiphyses."

Although many of the points in the clinical history of this and the two following fatal cases have been embodied in the foregoing part of the paper, yet in order to show the correspondence of certain morbid appearances with the clinical picture of the disease it is necessary that details of the history should be given with each fatal case, even at the expense of repetition.

Mr. Smith's case was a female child aged 1 year 11 months. She had been suckled for three months and subsequently fed on cow's milk and corn flour. There was no reason to think from the history that she had had congenital syphilis; but her rickets had been shown in her not cutting a tooth till she was twelve months old.

When thirteen months old, in April, 1874, her feet, legs, and thighs gradually became swollen and excessively tender, and, according to the mother's statement, 'hung down cold and dead.' This continued so for two months, and then there was slow improvement, so that by summer the swelling and tenderness had gone and the child was considered well, but for diarrhœa to which she had been subject more or less since six months old. At Christmas, 1874, the child being then twenty months old, the swelling appeared again in both lower limbs, first in the feet and legs then in the thighs. After a month the swelling of the feet had considerably diminished but that of the thighs had increased. She had sweated a great deal. The bowels were only opened twice or thrice daily and the motions were not offensive.

When admitted on Feb. 26th, 1875, that is, two months after the beginning of this second illness, she was an extremely pale and cachectic child. She was rickety with a large fontanelle and beaded ribs. She lay on her back with her lower limbs flaccid, never raising them. There was some œdema, more in the thighs than the legs, and scarcely any in the feet. The skin was natural in colour and not hot to the touch. There was some prominence in the region of the trochanters, the thighs projecting outwards there, more on the right than on the left side. On lifting up the limbs distinct softish crepitus was obtained without the slightest difficulty just below the hip-joint and knee-joint on either side. It was evidently produced below the epiphyses. The hip-joints and knee-joints seemed natural. There was no œdema elsewhere.

I regret not to have recorded a note at the time about the upper limbs. But a note taken a few days afterwards in the post-mortem room about them may be here intercalated, to the effect that there was crepitus obtainable below the head of each humerus but none in connection with the elbows or wrists. None of the epiphyses could be considered very large.

The child coughed a little and there was abundant shar

râle over both backs with some doubtful resonance at the bases. There was nothing abnormal to be detected in the heart or abdominal cavity. The urine could not be saved.

During the week she was in hospital the child lay on her back and whimpered a great deal, and continued extremely marasmic. She died rather suddenly, the cause being not perfectly obvious, but she was very feeble, and had, moreover, a considerable amount of bronchitis. Her temperature in the axilla had been as follows:

February 26th.—Evening 101·6°.
27th.—M. 100·6°. E. 100·6°.
28th.—M. 98. E. 100°.
March 1st.—M. 97·4°. E. 101·2°.
2nd.—M. 99·6°. E. 98·4°.
3rd.—M. 100°. E. 98°.
4th.—M. 99°.

At the post-mortem examination the lower limbs were first examined.

On the left side the glutæus maximus was found rather pale, but nothing else was noticed abnormal about it, or about the muscles attached round the head of the femur.

The vastus externus was swollen, pale, and pulpy. A little yellow serum bulged out of the upper part, and on cutting into the muscle some blood was found extravasated into its substance, and the same character applied to the vastus internus and crureus. The blood was pretty uniformly diffused through the deeper layers, and no coarse laceration was seen.

On making an incision down to the shaft of the femur, the periosteum was found separated from the shaft entirely in a continuous sheet. It was about $\frac{1}{8}$th inch thick, and was intensely injected all over the inner surface.

The shaft of the femur was separated from the epiphyses, and was almost entirely surrounded by a layer of maroon coloured blood clot, $\frac{1}{4}$ to $\frac{1}{2}$ inch thick, which loosely adhered to it, and in fact separated the shaft completely from the periosteum. On removing a little of this clot, the surface

of the shaft of the femur was seen to be perfectly smooth. The ends of the shaft were not splintered, but had a "sugary" surface. There was no lymph or suppuration anywhere.

The abruptness with which the changes were confined to the shaft was very striking.

The hip-joint was natural, and so were the lower epiphysis and knee-joint.

There was no extravasation of blood in the muscles of the leg.

The changes round the tibia were not so extensive as round the femur, but they were of the same character. The periosteum was thickened, vascular, and separated from the upper and lower thirds of the shaft by a thin layer of blood.

In the middle third there was no blood; the periosteum was adherent to the bone, but easily stripped up. The shaft did not, as the femur, lie absolutely separated from the epiphyses, but it was loosened at the upper end.

The left fibula at its upper extremity was natural; there was, however, separation of periosteum and effusion of blood to a slight extent at the junction of the lower epiphysis with the shaft. The ankle-joint was natural.

The right femur presented almost identical appearances with the left. There was a minute spot of blood in the floor of the acetabulum, but nothing else abnormal in the hip-joint.

The right tibia and fibula also presented almost identical characters with the corresponding bones of the left side.

The upper limbs were not examined beyond the ascertaining of the crepitus below the head of each humerus.

The abdominal organs were natural, and so was the heart.

There was some collapse in both lungs, and in part of the lower lobe of the left a small wedge-shaped patch of consolidation, very like a past pulmonary apoplexy, over which was a little velvety lymph.

As in this case, I regret to say I was responsible for

the suggestion of the term hæmorrhagic periostitis, there can be no impropriety in my now criticising it.

It was a provisional term employed to designate what seemed a remarkable and exceptional condition, but it would no doubt have been better to have described it as simply sub-periosteal hæmorrhage.

For there was, indeed, as is fully stated in the report, no proof of any true inflammatory process, and the appearances were, to a great extent, explainable by a primary hæmorrhage from the periosteum, the cause of that hæmorrhage, whether from damaged nutrition of capillaries, altered blood state, or both, being still an open question.

The second post-mortem was made on a female child, L. S—, æt. 10 months, who was brought to me as an out-patient in October, 1881.

She had never had breast milk, but had been fed first on condensed milk, then on cow's milk, and then on various foods; at the time when she was brought to the hospital she was taking Anglo-Swiss food. A teaspoonful of cod-liver oil had also been given to her thrice daily for two months. Careful inquiry failed to elicit anything in favour of syphilis, and subsequent examination of the other children was negative in this respect, although they were found extremely rickety. The mother was a delicate woman, but the father was healthy. The house seemed to be wholesome. This child had had much head sweating since she was three months old. Bowels had been constipated until two months ago, when she had a severe attack of diarrhœa, and after this her legs were noticed to be very tender. Three weeks ago her wrists also became very tender. When brought to the hospital she was extremely fretful. She not only screamed directly she was approached, and still more when she was examined, but continually whimpered as though in constant pain. Her axillary temperature was 99·8°. Her skin generally was pale to the last degree. There were ecchymoses in both upper eyelids; also underneath the mucous membrane

of the gums in the lower median incisor regions, and also in the lower molar regions separate ecchymoses were present. The child had not cut any teeth. The lower end of each radius was much enlarged, and the left hand hung prone in a condition of pseudo-paralysis. The left thigh was strongly flexed. There was some deep thickening to be felt along the lower third of the shaft of the left femur. The epiphyses of the lower limbs were a little enlarged. She lay on her back and never attempted to move. She was so ill that a complete examination could not be made, but it was ascertained that the liver and spleen were not enlarged. It was not expected that she would live, but the mother was ordered to give her as much raw meat juice as possible, and to continue the cod-liver oil. In a week's time she was brought again, and her condition was not worse, with the exception that there was slight proptosis of the left eyeball, as though there might have been some extravasation into the areolar tissue of the orbit. Eleven days afterwards this had subsided, but the child was paler and feebler, and she died October 29th, *i.e.* three weeks after she had been first seen, the total duration of her illness being about three months.

The post-mortem was made with considerable restrictions. Body wasted.

Cranium.—On the external surface of each parietal, just behind the fontanelle, there was found an area of thin sub-periosteal hæmorrhage, about the size of a shilling. The bone underneath it was slightly porous.

Thorax.—The intercostal muscles and some of the other muscles on the thoracic wall, especially the left serratus magnus, of a pale yellowish colour and slightly pulpy consistence as though infiltrated with serum. The periosteum of the ribs extensively detached, thickened, rather vascular, and slightly granular on the surface towards the rib, from which it was separated by a considerable quantity of flaky, chocolate coloured débris. There was no lymph or pus, and the flaky débris was more like disintegrated blood-

clot than anything else. The ribs were extensively bare and white and slightly rough. They were distinctly wasted. What had been taken for "beads" of the ribs were simply the extremities of the costal cartilages. The bony part of the ribs was much wasted especially along the anterior surface. Thus the anterior extremity of the rib was by no means in complete apposition with the whole of the extremity of the costal cartilage. There was no beading on the inner surface. The ribs were extremely brittle. They contained only thin soft medulla, and when this had escaped the rib was a mere shell. It was a wonder that the ribs had not separated at their junctions with the costal cartilages, or that some of them were not fractured beyond. They could be snapped in two easily. On the parietal pleura of both sides there were numerous petechiæ along lines corresponding with the ribs. There was a quantity of blood-stained serum in the left pleura but no lymph. In the middle of the left lung there were two or three very small masses of caseous tubercle and a few gray granulations on the surface in this neighbourhood. There was no disease of the right lung or pleura, and the bronchial glands were healthy. There was no disease of the other viscera.

We were not allowed to examine the lower limbs completely, but some extravasation was found partly into the periosteum near the left crista ilii and the deeper part of the muscles attached there, whilst the superficial parts were pale and slightly pulpy. There was also some subperiosteal hæmorrhage in the region of the junction of the upper epiphysis of the femur with its shaft.

The third post-mortem was also made on a female child, D. L—, who was an out-patient of my colleague's, Dr. David Lees, to whom I am indebted for ultimately transferring the case to me.

The child was not regarded as syphilitic by Dr. Lees or myself, but the following points of family history ought to be stated. The first child had been born dead at seven months. The second died aged four months, cause unknown;

the third was a full-time healthy child; the fourth was full time and suffered much from spasmodic croup and fits.

The mother stated that she was in good health during pregnancy with the child now in question, who was the fifth. The child was full time, is said to have snuffled occasionally but had no rash. She was suckled exclusively for seven months and not finally weaned till eleven months old. From seven months old, in addition to her mother's milk, she had cow's milk and two teaspoonfuls of Chapman's entire wheaten flour in the milk thrice daily; she never had condensed milk. At the time when the limbs became bad, *i.e.* at 17 months old, the mother states that she was giving her the best part of a breakfast-cupful of beef tea fresh-made every day with bread and farinaceous puddings and a little milk, not more than half a pint. No vegetables whatever were given to her. The mother affirms that the child had six teeth at six months. She was, however a weakly child from four months onwards. At nine months old she began to suffer from laryngismus, and she was first brought to the hospital on this account when 15 months old. She was decidedly rickety, had frequent diarrhœa, and when about 17 months old began to be very tender, especially about the lower limbs.

When she was admitted under my care, December 9th, 1881, she had had this extreme tenderness for two months, gradually becoming worse, so that she could not be touched anywhere except above the upper limbs without screaming. She was anæmic and flabby but not wasted, she sweated about the head considerably. The fontanelle measured 2 inches by 1; there was marked beading of the ribs, and the epiphyses of the upper limbs, especially at the wrists, were decidedly enlarged. There was some swelling over each scapula evidently belonging to the bone. On the right side it formed a low rounded tumour over the infraspinous fossa. Both were extremely tender to touch; there was no alteration of the overlying skin. Both thighs were swollen evidently from affection of the bone. The child was too sensitive to be examined thoroughly, but soft

crepitus was obtainable above and below both knees. The legs were also tender and swollen, less so than the thighs. The child was ordered beef-juice, mashed potatoes, and one orange daily. On the fourth day after admission she developed a clangy cough, and on the fifth a typical rash of measles, from the supervention of which disease she died on the sixth day.

At the post-mortem, examination of the limbs gave the following result :—There was considerable effusion of thin pale yellow serum into the substance of the muscles of the thigh, rendering the superficial layers pulpy. In the deeper layers there was tolerably uniformly disseminated blood-clot. The periosteum was thickened and, except at the upper extremity, separated from the shaft of the bone. Surrounding the shaft, and in some places bridging over the space between it and the displaced periosteum, was a thick sheath of blood-clot. There were no fluid contents in the periosteal sac nor was there any lymph or caseous material.

Along a line about ¼ in. above the junction line of shaft and lower epiphysis the shaft was separated. The separation had taken place through the loose imperfectly ossified material at the end of the shaft. The opposing surfaces were rough but not splintered. There was no callus. The ossifying centre of the lower epiphysis was a great deal of it diffluent. It was larger than natural, in fact, of the diffuse form which Mr. Sutton has recently pointed out as characterising the ossifying centre of rickety epiphyses. There was no cushion of cartilaginous material, such as is present in the early stage of rickets between the epiphysis and the shaft, but this, I take it, had been replaced by the loose imperfectly ossified material through which the fracture had taken place.

The muscles below the knee also showed the result of extensive blood extravasation in the deeper layers. The periosteum of the right tibia was thickened and vascular ; there was blood extravasation between it and the shaft for the whole length, but it was greater in amount at the

extremities than in the middle. There was fracture through the loose, imperfectly ossified, brittle material, about half an inch below the line of junction with the upper epiphysis. The ossifying centre of the upper epiphysis was diffuse, red, and very soft. There was no separation of the lower epiphysis from the shaft. The medulla of the shaft was very soft and red, and the trabecular structure of the shaft broke down very readily; there was no trace of suppuration or caseous material.

The periosteum of the right fibula was not actually detached from the shaft but it was very vascular, and there was slight extravasation between it and the bone, especially near the extremities.

The femur and tibia of the left side corresponded closely with those of the left side except that there was less extravasation.

The right scapula had a firm layer of blood-clot on both the ventral aspect and on the infraspinous fossa. That situated on the ventral aspect had led to the complete stripping of the periosteum, and the osteogenic power of the periosteum was shown by the formation of a thin lamina of osseous material over part of the subjacent blood-clot (*vide* fig. 2, Plate VII).

The clot on the infraspinous fossæ was also nearly $\frac{1}{4}$ in. thick, but I failed to detect any earthy material in the stripped up periosteum.

The right humerus did not present any periosteal lesions similar to those described above. It showed many of the features of rickets passing from the first to the second stage.

It is true the typical proliferated cartilaginous zone was no longer obvious as such, but its place was represented by a buff-coloured layer of imperfectly ossified material, and below this there was some loose trabecular bone.

The medulla was very red and soft and the trabecular structure of the shaft loose and scanty. There was no deformity of the shaft.

The radius and ulna were free from extravasation, and

presented on section rickety characters very like those of the humerus.

The lumbar vertebræ presented on section a marrow which was unduly red and soft and a trabecular structure which was very easily broken down. The ribs presented characteristic beads which were undergoing partial ossification.

The cranium did not present any bosses, but close to the medio-frontal suture and in front of the fontanelle was slight thickening, which was evidently old.

Some microscopic sections of the shaft of the femur, kindly made for me by Dr. Money, show the periosteum vascular and thickened, but I think without cellular infiltration; extensive hæmorrhage in the deeper portions and also between the periosteum and the bone; considerable absorption of the trabecular structure with large spaces showing in places slightly eroded margins; at the upper extremity rickety ossification.

There is a little to state about the viscera. The lungs showed many patches of collapse and some commencing lobular pneumonia. The bronchial glands were a little enlarged. The liver and heart were healthy. The spleen weighed nine drachms and was firm to the feel. There were some small, flat extravasations of blood under the capsule, and also some patches with a superficial area about the size of a shilling, of extravasation in the substance of the spleen, which, however, appeared to have caused but little laceration. The kidneys and intestines and peritoneum were natural.

We may now sum up the morbid appearances as far as these three cases are concerned, and add these to the general synopsis of symptoms.

Lower limbs.—Muscles: serum in the upper layer, which, probably partly as the result, are pale and slightly pulpy. Deeper layers contain extensively disseminated blood-clot.

Periosteum of femur and tibia thickened, vascular, separated from affected bones in great measure by sheath of blood-clot.

Fracture through loose trabecular structure at extremities (one or both) of shafts of femur and tibia. No callus. The two bony surfaces rough but not splintered.

I would suggest that the subperiosteal blood extravasation is the first event and the fracture the second. The extensive blood extravasation probably interferes with the nutrition of the bone, and thus the very minimum of violence, such as an ordinary movement, may lead to fracture.

The medulla of the shafts soft and red, and the trabecular structure scanty and friable.

Upper limbs.—Extensive blood extravasation under periosteum of both dorsal and ventral surfaces of scapula.

Unfortunately, with respect to the long bones, no complete post-mortem evidence is yet obtained, but there can be little doubt that in some of the cases that recovered there existed for a time a hæmorrhagic extravasation in the neighbourhood of the junction of shafts with epiphyses, especially near the wrists.

Ribs.—Extensive separation of periosteum, probably by blood-clot. Ribs wasted, bare, and brittle, with very thin red medulla. Ribs very readily separable from junctions with costal cartilages.

Cranium.—In one case subperiosteal hæmorrhages in position where Parrot's bosses are often found, and inasmuch as the scapula in one case showed new bone formed in the upraised periosteum, it seems possible that bone might also be formed over the subperiosteal hæmorrhages on the cranium and give rise to a condition indistinguishable from a cranial boss.

Visceral changes.—Blood-stained serous pleural effusion and petechiæ along the parietal pleuræ in the second case; hæmorrhage under the capsule and into the substance of the spleen in the third, and the small hæmorrhagic focus in one lung in the first, though perhaps not of great importance, are interesting from their association with the subperiosteal hæmorrhage.

(4) We must now ask what is the etiology of the disease under consideration, how is it to be distinguished from other diseases with which it has points of resemblance, and with what disease known to us has it the closest affinity.

Approximate answers to these questions will be best obtained by reviewing some of the conditions under which the symptoms arise.

As to age: My earliest case occurred in a child of five months, but Senator has described one in a child aged four months, and Steiner says the disease may occur as early as the fourth month. Of the thirty-one cases analysed (see Table, p. 208) twenty-six belonged to the first two years of life, and five to the second two years of life. Of the twenty-six cases belonging to the first two years, half belonged to the first year and half to the second. Dividing into periods of six months, the greatest number of cases occurred between six months and eighteen months of age, so that as far as our numbers help us the disease would appear to be pre-eminently a disease of the second part of infancy.

The sex is not stated in all the reported cases, but from the data forthcoming there appear to have been twelve males and eleven females, so that sex cannot be considered of any importance.

With respect to period of the year in which the symptoms become manifest,—Hirschsprung asserts that the disease always occurs in the winter months. This is much too sweeping a statement. Out of twenty-eight of which accurate data are given, seventeen occurred in the colder six months and eleven in the warmer six months. Probably a greater preponderance would appear if we had the dates of all the cases.

Although most of the cases belonged to the poor, there were several quite typical amongst children of those who were well to do, and there is nothing of importance to be elicited about the dwellings of the patients, which in some instances at all events were perfectly satisfactory.

It will be convenient to discuss the important question of diet at a later period, but possible hereditary causes may now be considered.

In several the mother was delicate, and in some of the cases other children were rickety in the ordinary sense, but in others the parents were healthy and the other children healthy. It does not appear from any of the histories that any other member of the family in any given case had suffered from the symptoms of so-called acute rickets, at least there is no statement to the effect that more than one member of the family suffered in this way, which is sufficient if not conclusive.

In regard to hereditary syphilis it must be admitted that it is difficult to prove a negative because the so-called acute rickets rarely develops during the period when the early indubitable syphilitic signs are present.

Steiner states that of the ten cases seen by him acute rickets supervened on congenital syphilis in two children aged four months, and that these two children soon died. As no post-mortem account is given of these cases it is open to us to ask whether the disease from which they suffered may not have been the congenital syphilitic affection of the ends of the shafts of the long bones which has been described by Wegner, Parrot, and others.

For, truth to say, this disease has some clinical features not unlike those which I have described as belonging to the so-called acute rickets, and it is desirable here to refer to the similarities and differences between the two.

The junction area between the shaft and epiphyses is specially affected in both, and in the syphilitic affection there may be some accompanying perichondritis and periostitis, which latter causes a swelling for a varying distance up the shaft. Also in the syphilitic disease there may be, as I have several times observed, displacements of epiphysis from shaft. Further, pseudo-paralysis, which is very common in the syphilitic disease, may occur especially about the wrists in the cases which occupy our attention. But in the syphilitic affection the pain and

tenderness are not nearly so severe as in the disease under consideration; they are often, indeed, quite trifling in amount, whilst in the so-called acute rickets they are more continuous and more severe than any bone disease of childhood with which I am acquainted.

There is occasionally a concomitant joint-effusion which may be purulent in the syphilitic affection. I think it is doubtful whether the joints themselves are affected in acute rickets. In a typical case of acute rickets in which the thigh is affected, the involvement of soft parts is more extensive than is ever met with in the syphilitic affection of the end of the shaft.

Finally, the element of age is of very great value.

Acute rickets, as I have shown, is very rare in early infancy, whilst the typical congenital syphilitic affection is common under six months, indeed, under four months, and even occurs in the fœtus. But it must be confessed the only thoroughly satisfactory distinction is one derived from post-mortem examination. In the syphilitic affection, of which I have examined three specimens post mortem, and of which specimens have been shown in this country by Mr. Haward and Dr. Goodhart, the change is mainly, as M. Parrot has pointed out, an endosteal one, and consists of what he calls a gelatiniform transformation of the ossiform material which exists at the extremity of the shaft. It is quite different in character from the massive proliferation of cartilaginous material found in an ordinary case of the first stage of rickets, which material forms a large cushion between the shaft and the epiphysis.

In the syphilitic affection, along with the gelatinous softening, there may be a varying amount of concomitant perichondritis and periostitis, which latter, as I have said, may extend up the shaft for a varying amount and be followed by an osseous deposit. But I have not seen in the syphilitic affection the extensive separation of periosteum and shaft by a mass of blood-clot such as occurs in the specimens of so-called acute rickets now under consideration, and such a striking condition has not been described by

Wegner or Parrot who have both examined a great many cases.

But the affection of the ends of the shafts is by no means the only form of syphilitic affection of the long bones. Cases may be seen in older children where several long bones are thickened along the greater part of the shaft with firm solid deposit. This condition may last for months and slowly clear up. How is this to be distinguished clinically from acute rickets? I can only say that I have never seen a syphilitic case of this character lying prostrate with the pain, tenderness, and cachexia comparable with that we have already considered, but that in fact the general suffering and the progress are quite different.

To return to the cases analysed in this paper. In the greater number, as I have said, the early indubitable signs of congenital syphilis are no longer capable of being brought into evidence on account of the age of the children, but are there any other signs available which might help us in this direction? In no less than eight cases either the facial or cranial bones were affected, and in two if not three there were bosses on the frontals or parietals of the kind described by M. Parrot as characteristic of congenital syphilis. Now, although it is certainly the fact that many unquestionable syphilitic infants develop these bosses, especially if they be also rickety, yet in our present state of knowledge it seems to me premature to regard them as decisive of the question of congenital syphilis in the absence of other signs.

One of my cases, along with these cranial bosses had marked splenic enlargement, and both these conditions persisted after the affection of the limbs and the sponginess of the gums had subsided, not under mercurial or iodide treatment, but under raw-meat juice and vegetables. In this case I have unfortunately lost the early history; the conclusion I had formed was that the child was possibly syphilitic, but that this had nothing to do with the condition of the limbs.

The early infantile history of one of the three cases of which post-mortem notes have been given, was compatible with congenital syphilis, but there was nothing conclusively syphilitic found post mortem.

It must be remembered that although the effects of syphilis may last for a considerable time, and seem to have a very special incidence upon and proneness to relapse in the osseous system, yet that they may also very rapidly pass away. Thus I made a post-mortem examination on a child aged ten months, who died of acute tuberculosis without any syphilitic lesion, and whom I had had under observation when it was an infant with severe congenital syphilis. There must be many parallel experiences.

To sum up these observations it may be stated—(1) that of the cases recorded in the great majority there was no conclusive proof that congenital syphilis was actively present; (2) that in several there was nothing in the previous history to justify the view that congenital syphilis had been present in early infancy; (3) that even in those in whom infantile syphilis had possibly existed it would not necessarily follow that the symptoms of acute rickets had any connection whatever with the infantile syphilis; (4) that it seems possible that two cases, briefly mentioned by Steiner, of children four months old, who were the subjects of congenital syphilis, and considered by him to be also suffering from acute rickets, were really the subjects of the special syphilitic affection of the ends of the shafts of the long bones, which presents considerable resemblances to the so-called acute rickets.

We must now consider the relation of the disease in question to the ordinary form of rickets with which we are so familiar.

Was rickets present at all in the ordinary acceptation of the term? In nine cases the details are not sufficient to allow us to give a definite reply. Of the twenty-two remaining cases reference to the table will, I think, show that in at least three the signs of ordinary rickets were

very pronounced indeed, in seven moderately well marked, and in nine slight.

In at least three we are justified in saying that there was no rickets, and amongst the nineteen slight cases the evidence often amounts to nothing more than slight beading of ribs.

Hirschsprung asserts that the disease has always appeared in formerly healthy children. This is another of the sweeping statements of a very able observer. But, although the statement is not accurate, yet it is remarkable that many of the children before the sudden onset of the disease were considered to be in fair general nutrition.

In Dr. Fürst's case, after the subsidence of the acute symptoms, marked bendings of the femora were found which had not occurred before the illness, and which he considers established the truly rickety nature of the illness.

I believe that even that condition is susceptible of another explanation, but whether that be so or not let it be noted that in several of the recorded cases the recovery was absolute without deformity within a period of three or four months, which is quite unlike the ordinary course of rickets.

We are confronted then by two difficulties:—1st. The complex of symptoms which we have described may occur in a child in whom the ordinary signs of rickets are practically nil; and 2nd, very severe cases of rickets, in the ordinary sense, may run their course without presenting the complex of symptoms which we have described.

There is little wonder then that the German writers have found it difficult to "dovetail" the so-called *acute rickets* with ordinary rickets, the more so that it is admitted that acute rickets so called is quite a different thing from severe or aggravated rickets.

I do not think that anybody will maintain that the subperiosteal hæmorrhage in the cases of which the post-mortems have been given (and which also I assume to

have been present in the cases analysed) is a feature of rickets as such.

I showed the specimens to Sir William Jenner, who told me that, in respect to the striking feature, viz. the subperiosteal hæmorrhage, he had never seen it in ordinary rickets. There is also no description of such a condition in Guérin's account of rickets.

With respect to other diseases, I have already shown that, clinically and anatomically, the one in question differs from acute periostitis single or multiple.

The partial death of the shafts in severe cases is, I think, sufficiently explained by the mechanical interference with efficient vascular supply by the extravasated blood clot. In the cases which recover, the thickening of the bone shafts, which may remain for a considerable period, may, I think, be explained by the osteogenic power of the upraised periosteum.

With what disease can we connect this non-inflammatory, sub-periosteal hæmorrhage, associated with blood in the deeper muscular layers and serum in the superficial layers? Blood has been found in the joints in hæmophilia, but it is not described as occurring under the periosteum in that disease.

In the group of cases analysed there is, I think, no proof of effusion into the joints, although once or twice the term "painful joint affection" is used. In a great many, at all events, there is no sign of effusion. But certainly the clinical history of our group is very different from that of one of the arthritic attacks of hæmophilia.

It is a question how far purpura ought to be ranked as a separate disease, and whether it would not be desirable to consider it rather as a symptom occurring in many diseases. Although sugillations occurred a few times in our present group it is interesting to note that only in one case (XXIII) are small, numerous, spotty ecchymoses in the skin recorded. Without attempting to define purpura hæmorrhagica, I may mention that in a few cases (to which for want of a better this term might have been

applied) I have seen general painful swelling of the leg occur along with the appearance of purpuric spots in that region. This painful swelling has, however, so far as I have seen, only lasted a few days, and thus differs considerably from what obtains from our present group.

Although it would be absurd to limit the possible occurrence of sub-periosteal hæmorrhage to any one malady, I believe it will be found, on analysis of the cases before us, that they approximate more closely to scurvy than to any other disease with which we are acquainted. Let us consider the parallelism first along anatomical lines.

The painful brawny induration of the lower limbs in adult scurvy was shown by Lind to be due to blood extravasation in the bellies of the muscles and serum in the tunica adiposa (p. 496, 3rd edit.). He often found the blood extravasation most extensive in the deeper layers, and lying on the periosteum; and once he discovered it lying in spoonfuls beneath the periosteum.

Dr. Budd, in his article on "Scurvy," in Tweedie's 'System of Medicine,' describes a post-mortem on one case in which, although there was no swelling of the calf, there was a node-like swelling over one tibia, and on cutting down upon it there was found a thin layer of blood under the fascia and a solid clot of chocolate colour a line or two in thickness for a length of six or seven inches under the periosteum, the periosteum itself being thickened and infiltrated with blood in this region. Other sub-periosteal hæmorrhages were found on one femur, one fibula, the opposite tibia, and the upper and lower jaws.

There are some older observations which are still more interesting. In the year 1699 M. Poupart made some dissections of scorbutic bodies in the Hospital of St. Lewis, at Paris. Amongst his remarks is the following, quoted by Lind:—"In some, when moved, he heard a small grating of the bones. Upon opening those bodies the epiphyses were found entirely separated from the bones, which by rubbing against each other occasioned

this noise." All the young persons under eighteen had in some degree their epiphyses separated, and "in some" he says "we perceived a small low noise when they breathed," and in them the cartilages of the sternum were found separated from the bony part of the ribs.

He further describes a condition of rib very like that to which I have referred in my second post-mortem.

There is another observation by Dr. Godechen, a Russian physician, which is quoted by Budd, and is very important. In a case of scurvy, separation of the ribs from the costal cartilages and fractures of ribs near their anterior extremities occurred, without violence, whilst the patient was in the hospital.

Without laying too much stress upon it, I may refer to the blood-stained effusion in one pleura in my second post-mortem as being comparable with the condition of the pleura found in some of the fatal cases of adult scurvy.

Having shown, then, a certain anatomical resemblance between our group of cases and adult scurvy, let us work back along the clinical lines and see how far they also run parallel.

The order of appearance of symptoms, as set down by Lind in his own words, is, first "a change of colour of the face, from the natural and usual look, to a pale and bloated complexion with a listlessness to action." The second symptom is a stiffness and feebleness of the knees upon using exercise, and the third is the swelling of the gums.

In our group of cases the change of colour of the face is most striking. In a typical case it is not simple pallor but pallor of a somewhat dirty sallow tint. The general prostration is quite as marked a symptom. With regard to the swelling of the limbs during life, though it is true it is not so brawny in our cases as in the adults yet there are many similiarities. The distribution is singularly parallel. As pointed out by Lind, there may be in the adult only a single swelling, but more commonly the swelling is bilateral or indeed multiple; and this obtains also with

regard to our cases. In both alike the lower limbs are in the majority of cases affected and in the greatest severity.

There are parallels also in distribution, to which I have already referred in the occasional involvement of the ribs and of the upper and lower jaws.

With respect to the gums, we have, it would seem, at first a remarkable divergence between some of our cases and the typical adult scurvy.

In fifteen cases out of thirty-one it is noted that the gums were affected. In a few of these the swelling was obvious and characteristic, as in Dr. Cheadle's two cases, especially the first, from which there was much bleeding. Also in the case of Dr. Ingerslev the sponginess of gums was accompanied by a carrion like odour, which was no doubt sufficiently suggestive to him of the true affinities of the malady. But in several others the swelling was very slight, and in fact only consisted in small localised ecchymoses in the sites to be occupied by the coming teeth. In six cases it is specifically stated that there was no stomatitis, and in ten it is not mentioned as being present.

The question of scurvy was indeed considered at the time of making the post-mortem of Mr. Thomas Smith's case, and dismissed on account of the absence of any swelling of gums.

But even in adults I have, since making the above post-mortem, learned that the absence of swelling of the gums does not negative scurvy. Dr. Ralfe has informed me that in crews suffering from scurvy there have been well accredited cases of men who have had all the other symptoms of profound cachexia, &c., but without the spongy gums; and this view is also expressed by Dr. Buzzard in his article on scurvy in 'Reynolds' System,' and in his definition he implies that sponginess of gums is not absolutely essential. I believe it has been observed that if a man who has lost all his teeth gets scurvy subsequently, sponginess of gums does not occur. Sir James Paget has told me that it is almost impossible to salivate a patient who has lost his teeth, and

the difficulty of inducing sponginess of gums in young infants by the administration of mercury is well known. It is interesting to note in our own group that where no eruption of teeth had occurred no sponginess occurred; that the sponginess when present was chiefly in the neighbourhood of teeth that had been cut, and that the small submucous ecchymoses when present were above the sites of the oncoming teeth. I submit, then, that this divergence is not sufficient to disprove the identification of the so-called acute rickets with scurvy. The history of the study of disease has led us to discredit universal propositions in medicine and to doubt the existence of an absolutely pathognomonic sign, that is to say, of a sign which is present in every case of a given disease and never present in any other disease.

To those who would be willing to admit that the cases with limb affection and spongy gums were truly scorbutic, whilst denying the scorbutic character of the cases of limb affection without spongy gums, I can only reply that in every other symptom several of these cases were as nearly as possible identical.

With respect to pyrexia there is another seeming divergence. I have already pointed out the inaccuracy of Senator's statement that in these cases some fever is an invariable and characteristic symptom, and I have suggested that the fever, when present and not due to intercurrent ailments, may perhaps be proportionate to the tension of the hæmorrhagic effusion under the tight periosteum. The bones have not been carefully examined in a sufficient number of cases in adult scurvy to admit of a dogmatic statement, but it seems probable that in them the periosteum is not so extensively involved as in these children's cases; that in fact the blood extravasation and serous exudation are more superficial and may give rise to less tension. On the other hand, although scurvy in adults is generally an apyrexial disease, it must be remembered that, in the words of Dr. Budd, occasionally we find the skin hot and the pulse attaining or even

exceeding the rate of 120 in the minute. In these cases, Dr. Budd remarks, the swellings are exquisitely tender and the slightest movement of the limbs occasions great suffering.

Thus, even in this respect, I think we may establish a certain parallelism.

There are many other parallelisms to be drawn from a further analysis, but we must no longer defer the consideration of diet as an etiological factor of these cases. The great difficulty in this part of the inquiry is the paucity of information in many of the reports as to the quantity and quality of the food which was being taken at the time of the onset of the acute symptoms.

First, with respect to breast milk. Several of these thirty-one children had previously been suckled for varying periods, but with the exception of a dubious statement by Möller about his third case, I think none of them are recorded as being at the breast at the period of onset of the acute symptoms. Steiner, however, in his brief account of the disease, though he speaks of its onset as being generally after weaning, uses the phrase that it may even appear during lactation. I venture to suggest that these exceptional cases coming on during lactation may have been not the so-called acute rickets, but examples of the congenital syphilitic bone disease which may appear in in infants at the breast, if the subjects of syphilis.

In the cases at present under review, then, we have to deal with children fed at the time and for a varying period previously by hand, and it is of the greatest importance to see if there was any other point in common with respect to the food.

First, it will be found that certainly five, probably six, and perhaps more, were taking cow's milk at the time of onset. On further investigation, we find that in one case the quantity was extremely small, in another not more than half a pint daily; in a third it was two pints in twenty-four hours, with an equal quantity of water, the child suffering the while from considerable diarrhœa.

We have no information about the quantity in the other cases nor indeed of the quality, which is probably important.

It is clear that the use of beef tea was not adequate to prevent the appearance of the disease, for in three cases, perhaps four, this food was being given at the time of onset.

It is very important to ascertain whether the affection ever appears whilst a child is taking raw-meat juice. The only case bearing on it is Förster's, but it is not explicit enough.

A child of eleven to twelve months old had been breast-fed for three months, then had had cow's milk, Liebig's soup, flesh broth, eggs, scraped meat, &c., and had suffered from jaundice and diarrhœa. But it is unfortunately not clear from the account of the case what was the exact diet at the time of onset. This is important, because for several months the child had been well nourished in spite of the jaundice and diarrhœa; and Förster notes that the evidence of rickets was very slight.

Two of my cases had hysterical objections to meat and meat and vegetables respectively. One of them cried and even vomited when I had a plate of meat set before him. Also in two cases of scurvy in older children not included in this analysis I have observed the same curious dislike. One of these latter children absolutely screamed whenever any vegetables were placed on the table anywhere near to her, and refused any kind of food offered to her if the spoon had been previously used for vegetables.

I cannot find that it is stated in any of these cases that at the time of onset fresh vegetables formed a part of the diet.

The most important fact is that in at least seven there was absolutely no fresh food given. Thus Nestlé's food made with water, Ridge's food made with water, mealy foods, exclusively amylaceous food, and Anglo-Swiss food

are examples.* In some of the cases the food and hygienic conditions are said to be satisfactory, but as the details are not given we cannot discuss them.

What light does the result of treatment throw upon the disease? Möller's cases convinced him that anti-phlogistic remedies were distinctly injurious, and in Dr. Fürst's case they seem not to have been followed by any benefit.

Great influence is attributed by Bohn and Hirschsprung to the return of spring and the possibility of getting the child out, and this is parallel to the experience with regard to adult scurvy.

With regard to antiscorbutics, they appear to have been given in some of the German cases without, in the opinion of the authors, leading to obvious benefit.

I venture to suggest that, before arriving at a definite conclusion, it is necessary to know the exact period of the disease at which they were given. When the marasmus is very profound indeed, it is, perhaps, too much to expect an immediate improvement, or, perhaps, any improvement at all. And, moreover, when there is much sub-periosteal hæmorrhage, it must take a considerable period before absorption can possibly be completed, and the bone return to a normal state. But in Dr. Cheadle's two cases, and in seven of my own, viz. xvii, xx, xxi, xxiv, xxvii, xxviii, xxix, the improvement was perfectly obvious and most striking in those which could be personally supervised, and in which treatment could be persevered in, in spite, as sometimes happened, of objections on the part of the child.

In Ingerslev's case, it is noted that no treatment, including antiscorbutics, led to the slightest improvement until the spring came, and the child was able to eat garden cress. This is parallel with an interesting observation recorded by Dr. de Mertans in the 'Philosophical Transactions' for 1778. This physician, who was attached

* To which I may add, from two subsequent cases not included in this analysis, Savory and Moore's food and Neave's food.

to the Foundling Hospital at St. Petersburg, had been accustomed to treat many severe cases of scurvy in children, especially in the winter and spring, and a very fatal disease he sometimes found it. Experience taught him that if the cases came under his care early in the disease vegetable soups succeeded very well, so that three or four weeks were generally sufficient for cure. But in one winter the outbreak was particularly severe and resisted his ordinary treatment, and he then found that his most stubborn cases yielded only when he gave them raw vegetables as well as vegetable soups ('Phil. Trans.,' vol. lxviii, p. 676).

Reverting to our cases it is clear that they differ very much in severity. Those in which the cachexia is very profound often end fatally, just as in adult scurvy. Nevertheless, a careful perusal, especially of the German cases, convinces me that the disease in question often tends towards a slow, but ultimately complete, recovery, and this independent of any special treatment.

Let us turn once more for a parallel in adult scurvy to Lind, whose work is such a masterpiece, not only of learning, but of accurate and candid observation. Lind's arguments for the employment of fresh vegetables in scurvy are unanswerable, but it is interesting to read in the post-script to his third edition, concerning certain cases that he had carefully watched, that "the strict abstinence from the fruits of the earth was continued long enough to convince me that the disease would often, from various circumstances, take a favorable turn, which cannot be ascribed to any diet, medicine, or regimen whatever."

It may very properly be asked why, if it be true that these cases are mainly produced by a faulty diet, are they not more frequently seen, since a faulty dietary must obtain in London and other large towns to an extreme degree, especially amongst the poor?

A complete answer to this question cannot be given. But first, probably minor, degrees of scurvy are not so rare as might be thought.

It is possible that some slight cases, and even severe cases of the bone affection, are dismissed as ordinary rickets, with an excess of tenderness and fretfulness. Probably, also, in this affection as in others, idiosyncrasy plays a part; and we have to remember that in adults the scurvy-producing diet may be in use for a considerable time before the disease is precipitated, so to speak, by some additional, often unknown, depressant agency.

A valuable remark of Dr. Cheadle's may, however, be referred to in regard to the reason why scurvy does not more often occur amongst the children of the London poor. A bread and butter diet, with the exclusion, or extremely meagre supply, of milk is common enough, and is probably responsible for a great deal of rickets, but poor children are often saved from scurvy by the common use of potatoes. If potatoes are excluded and only the bread and butter diet given scurvy, sooner or later, is exceedingly likely to manifest itself.

To sum up this paper, I will submit that (1) the characteristic symptoms of the so-called acute rickets, viz. the special limb affection and the cachexia, with or without sponginess of gums, are not due to rickets at all but are truly scorbutic.

(2) That the anatomical basis of the limb affection is sub-periosteal hæmorrhage, and that this hæmorrhage probably accounts for some of the anæmia.

(3) That the disease may occur in rickety children, and perhaps in them more readily than in non-rickety children, but that the amount of rickets may be almost nil.

(4) That although the disease tends spontaneously in many cases towards a slow but complete recovery, marked improvement often follows a vigorous and especially an early antiscorbutic treatment.

(5) That the treatment recommended is.—*locally*, during the acute stage wet compresses and avoidance of movement, at a later period careful shampooing and douches; *internally*, the use of raw-meat juice, fresh milk,

and orange juice, or of some other fresh raw vegetable, and from the first the access of as much free air as is possible.

(6) That the use of the term acute rickets should be abolished for these cases, and that of infantile scurvy substituted, the special note of which, as distinguished from adult scurvy, being the greater incidence of the disease on the bones.

(7) That in regard to the hand feeding of infants it seems probable that the so-called "infant foods" cannot be trusted as sole aliment for any lengthened period, however useful they may be as adjuncts.

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Addenda.

(1) I have been favoured by my friend Dr. Stephen Mackenzie with a memorandum on the post-mortem examination of an infant who during life presented clinical features resembling those described in the text.

Autopsy (October 30th, 1878).—Throughout the intestines, as they lay in the abdominal cavity, were seen numerous patches of red and purple red colour showing through the peritoneal coat. In the large intestine all of these were small and round, in the small intestine they varied in size and shape, but the majority were much larger, mostly oval, with their long axis corresponding to the channel of the intestine. On closer examination it appeared that this red colour was chiefly beneath, but slightly involved, the peritoneal coat. On opening the intestine it was found that each of the patches occupied solitary glands. In the large intestine, where solitary glands are discrete, the patches were small; in the small intestine the aggregation of the solitary glands into the oval Peyer's patches gave rise to the round and oval patches of extravasation. There is no doubt that the patches are extravasations into diseased lymphatic structures. The mesentery showed small raised bodies, looking like miliary tubercles, but of red instead of grey colour. The lungs were spotted over on their surfaces and in their interior with hæmorrhages varying in size and shape, not very dense in consistence.

One kidney showed hæmorrhages almost exclusively affecting the pyramidal structure.

At the anterior extremity of the ribs, almost black elevations or nodules were seen; in some cases a similar black appearance was seen extending along the

course of the rib for some distance. The nodules felt hard on cutting into them. The rib was found rough, completely stripped of its periosteum, and there was a quantity of blood between the rib and the periosteum. The changes appeared to begin at the junction of the ribs with the costal cartilages. Many ribs were affected in this way.

The shaft of the left femur and tibia were examined; the bones were found completely stripped of their periosteum, rough, and lying in a cavity, which was filled with blood. The epiphyses were separated from the shafts; the bones were red, and so was the medullary canal; the periosteum deeply blood stained.

(2) For further anatomical evidence I may refer to a paper by Mr. Page, which appears in the current volume of the 'Transactions,' entitled "Subperiosteal Hæmorrhage, probably Scorbutic, of Three Long Bones in a Rickety Infant." In this case incisions were made down to the bone and extensive blood-clot removed. The child under a change of diet made a complete recovery.

(3) Mr. Gardiner, of Dulwich, has supplied me with notes of a case of a child two years old, who was under his care with affection of the lower limbs similar to that described in the text, and also with spongy bleeding gums. The child had been fed on cow's milk and Savory and Moore's food, but the quantity of the former had become reduced to about three quarters of a pint in twenty-four hours, and even to seven or eight ounces. Treatment for over a month was unavailing until the measures suggested in the typical case were adopted; these were followed by the subsidence of the gum condition within forty-eight hours, and of almost all the pain and swelling of the limbs within thirteen days, leaving a little thickening of part of one tibia, which also disappeared.

(4) My colleague, Mr. R. J. Godlee, has informed me of

a case of this nature recently under his care, which he proposes to publish in detail. The patient was a rickety child of eleven months old, who, after a severe attack of diarrhœa, began to suffer from painful swellings of the lower limbs like those described in the text. Separation of the lower epiphysis of each femur and of both epiphyses of one tibia was found. Some subconjunctival hæmorrhages had occurred, but there was no sponginess of the gums. There was profound anæmia. After seven weeks' severe illness, going from bad to worse, the child made a sudden improvement under a modification of diet to which lemon-juice was added. In little more than a fortnight the epiphyses had become reunited. No local treatment was employed except sand-bags.

(*September*, 1883.)

Table of Cases

No. of case and name of observer.	Age; sex; period of year.	Lower limbs.	Upper limbs.	Skull and other bones.	Gums.
Nos. I, II, III. (Three cases) Möller, 1856-60	From 1 to 3 years	Lower ends of the femora and of the bones of the leg; great tenderness	Upper end of the right humerus swollen, and the forearm bones	—	—
*No. IV.					
(Fifth case) Möller	21 months, March	Illness began with pain, referred to right ankle after a fall. Nothing at first could be detected wrong; then pains referred to all the joints of the lower limbs. Six weeks after onset, came swellings about ankles, knees, forearm bones, and ribs, simultaneously with great tenderness and immobility. After the end of the illness marked increase in length growth of the lower limbs was observed	Pain and tenderness appeared in upper extremities after lower limbs and back; ends of the bones of the forearms became swollen simultaneously with swelling of lower limbs	Tenderness in the back and neck; swellings at the ends of the ribs	Swollen in the course of the illness, about 2½ months after the onset, and simultaneously with appearance of sugillations on one leg
No. V. Bohn, 1868	Two attacks: first when 16 months old, in March; second when 28 months, in Feb.	"Ankle and knee-joints swollen and painful on movement; thighs and legs kept flexed"	—	Head "large and swollen"	Ulcerative stomatitis
No. VI. Förster, 1868	11 months, male, December	Swelling of diaphysis of both femora, with slight enlargement of the extremities; some swelling of upper part of both legs; great tenderness on movement. Increased length noticed after recovery	—	—	Stomatitis, especially around the recently cut teeth, simultaneously with affections of limbs
No. VII. Hirschsprung, 1872	16 months, female, March	Left shin and foot, followed by swelling of the right foot, which left a little thickening of the right ankle	Lower end of left radius; upper end of left humerus	Swelling of upper and lower jaws, which swelling relapsed	Swelling of gums and lower lip, which appeared some days before the swelling of the limbs
No. VIII. Senator, 1873	4 months, January	Both femora, tibiæ, and fibulæ; tender swellings near epiphyses; no redness	Both humeri, radii, and ulnæ; tender swellings near epiphyses	—	—
No. IX. Ingersley, 1873	1 months, male, winter	"Painful joint affection"	—	—	Spongy gums; odour of carrion

* Möller's fourth case (related in the text) is a doubtful one, and is therefore not included in the table. No. IV of *this table is Möller's fifth* case.

DESCRIBED AS ACUTE RICKETS.

Skin, temperature, and general nutrition.	Heredity.	Previous health and signs of ordinary rickets.	Food and dwelling.	Progress and duration.
Temperature over swellings at first elevated, afterwards normal. No fever. Extreme anæmia	Mother of one case decrepit and tuberculous	—	Two were fed with "mealy food;" diet of the other not given	Two completely recovered without deformity after many weeks. One died of atrophy, the lower ends of the leg bones being still thickened and tender. Anti-phlogistic treatment was injurious. The application of three leeches to the knee in one case followed by profound anæmia and involvement of the other knee.
Temperature not elevated in early part of illness; about 2½—3 mos. after onset febrile with intestinal and bronchial catarrh. Extreme anæmia. On one leg there appeared some sugillations about 2½—3 months after onset. Atrophy of muscles. Urine: no excess of phosphates, considerable deposit of uric acid	Mother tuberculous	Suckled by a healthy wetnurse; had been well developed and strong up to the time of weaning; suffered little with teething. At the end of the present illness there was severe head-sweating	Not definitely stated what food was being taken at the time of onset; considered satisfactory	Total duration about 4½ months. During the early period cod-liver oil was taken. The application of four leeches to neighbourhood of one trochanter early in the disease was followed by profound anæmia. At a subsequent period quinine, phosphoric acid, iron, lime-water, &c., were successively tried, and, finally, limejuice and fresh vegetables. The writer states that all these were given without the least benefit. Rapid improvement followed the setting in of warm weather, when the child could be taken out into the fresh air all day. [Let it be noted that the anti-scorbutic treatment had been started a short time before improvement set in.]
Extreme cachexia, not febrile	—	During illness much sweating about upper part of body. Had had pneumonia and diarrhœa at 11 months	Hand-fed after 2 months. Dwelling dark and damp	Recovery from first attack in 5 or 6 months. The second attack lasted over 3 months.
—	—	Had been considered healthy in spite of having had jaundice and diarrhœa; characteristic rickety shape of thorax, but this only slight in amount. First tooth at 6 months	On cow's milk, Liebig's soup, flesh broth, eggs, and fat	Recovered completely in 2½ months on cow's milk, cod-liver oil, and malt, and sea-salt baths.
"Skin bluish-red in several places." Pyrexia, anæmia, emaciation, vomiting, diarrhœa. Urine normal	Brothers and sisters healthy; none of them rickety	Previous good health; fontanelle closed; 12 teeth; could walk	Had been formerly breast-fed; details not given of food at period of onset of symptoms. "Healthy conditions"	Treatment ineffectual until month of May, when she was able to be carried out, and then there was rapid recovery. Duration probably 3 months.
Febrile	—	Said to be previously healthy and well developed	Nestlé's food	Duration 6 months.
Anæmia, wasting; no hæmorrhage; no intestinal catarrh	Healthy parents	—	At time of onset of symptoms fed only on amylacea. Lived in the country	Duration nearly 6 months. No improvement on iron, quinine, and anti-scorbutics, until spring, when child got abundance of garden cress, and recovered completely.

No. of case and name of observer.	Age; sex; period of year.	Lower limbs.	Upper limbs.	Skull and other bones.	Gums.
No. X. Petrone, 1881	11 months	Painful swelling of lower epiphyses, of both femora, and both tibiæ, and both fibulæ; the joints natural; great tenderness on movement	Painful swellings of lower epiphyses of both radii and ulnæ	—	—
No. XI. Fürst, 1882	25 months, female, February	Cylindrical swelling of left thigh and leg, and of right leg. In a relapse right thigh became also affected; skin tensely stretched, reddish colour; extreme tenderness; flexed. After all the acute symptoms were over, a sharp angular bending of the femora was found, besides massive thickening. No marked increase in length occurred	Swelling and redness of lower half of right forearm occurred subsequently to the swellings of the lower limbs; after this the right arm became swollen, red, and painful, and then the left arm and left forearm became swollen near the lower epiphysial region	During relapse occiput became tender, and some thickening was found in the zygomatic regions	No stomatitis
No. XII. Dr. Gee, Case 1, 1881	14 months, October	Lower half of right femur and right tibia swollen and painful	—	—	—
No. XIII. Dr. Gee, Case 2	7½ months, male, June	Both femora thickened and tender	Scapulæ thickened; upper end of both humeri; carpal end of both radii enlarged	Back weak; considerable bending in of ribs outside the nipple line; sternum recedes. Deformity unlike that of rickets	—
No. XIV. Dr. Gee, Case 3	12 months, female, May	Both tibiæ swollen for 2 inches upwards from the lower end and tender. Subsequently lower half of right femur became swollen	Both shoulders became swollen	Swellings on frontal on each side of the fontanelle, like Parrot's swellings	Ecchymosis gums where t are being cut
No. XV. Dr. Gee, Case 4	16 months, female, May	Lower end of right tibia swollen and tender	—	—	—
No. XVI. Dr. Gee, Case 5	8 months, male, August	Lower half of left tibia enlarged; probably very painful, especially at night	Lower end of right radius enlarged	—	—
No. XVII. Dr. Cheadle, Case 1, 'Lancet,' Nov. 16, 1878	13 months, male, January	Thighs and legs swollen; thickening of femora felt; extreme tenderness all over. Four months ago had a blow on thigh, which left a swelling for some considerable time	—	—	Spongy, mor less, 3 mo before limbs noticed. G have bled a g deal

Skin, temperature, and general nutrition.	Heredity.	Previous health and signs of ordinary rickets.	Food and dwelling.	Progress and duration.
Some pyrexia. Slight swelling of spleen and lymphatic glands. Appetite and digestion normal	Father was healthy, but had been rickety in childhood	Previously strong and well nourished. Suffered during this illness with head-sweating	—	One month. Was treated with quinine, aq. calcis, and strong nourishment.
Pyrexia, cachexia	Parents healthy (neither syphilitic nor scrofulous). Father had traces of early rickets. This the first child	Healthy in first six months of life. First tooth at 12 months. Laryngismus stridulus. Enlarged epiphyses for at least 10 months before the acute symptoms appeared	Details not given as to food which was given at the exact period of onset of acute symptoms; for first 6 months goat's milk, cow's milk, and cocoa; subsequently rusks and soup. Present abode dry and sunny, but former abode damp	Duration 2½ months. Calomel internally, lead lotion and iodine externally, were followed by a very slight and transient improvement. Treatment generally appears to have had little effect.
Extreme anæmia; no pyrexia. No signs of internal disease, except slight bronchitis	—	—	—	2 months. Fatal.
No pyrexia; deep cachexia. Small lump showing dark through skin in lower dorsal region	Several deaths from phthisis in mother's family. This the fifth child; others healthy	Healthy till 7½ mos old, when he began to pine and suffer from pains in limbs. Costal ends of ribs not much enlarged; fontanelle nearly closed; has 6 teeth	—	4½ months. Fatal.
Hæmorrhage into left eyelid; bruise over sternum; deep cachexia. Vaccination wounds did not heal well and left large scars	Father had had gonorrhœa, but denied having had a chancre. This the fourth child; no miscarriages or still-born children	Moderate beading of ribs	—	About 4½ months. Fatal.
Much emaciated	This the fifth child born alive; miscarriage between third and fourth, also between fourth and fifth	Ribs rickety	—	—
No pyrexia; great cachexia; hæmaturia for a few weeks	—	Never healthy; had had eczema once, leaving a few scars; ribs much beaded. Has cut the two lower incisors	Hand-fed	10 weeks. Treated with cod-liver oil, iodide of potassium, and Parrish's syrup.
Muddy complexion. Temperature mostly normal or subnormal, but on day of admission rectal temperature was 108°. Slight albuminuria for a few days. Two unhealthy sores, one on right wrist, one on forefinger	Mother phthisical; father healthy; four other children weakly; none have suffered like this.	Large fontanelle	1 quart of milk daily till 10 months, then bread, Ridge's food, and small quantity of milk; would not take gravy or potatoes	2 to 3 months on admission. Began to improve on anti-scorbutic diet. After the swelling of the legs had subsided there was some desquamation. Thickening had quite disappeared in 6 weeks. Total duration about 4 months.

No. of case and name of observer.	Age; sex; period of year.	Lower limbs.	Upper limbs.	Skull and other bones.	Gums.
No. XVIII. Dr. Cheadle, Case 2, 'Lancet,' July 15, 1882	10 months, male, November	Swelling of both tibiæ, right more than left	—	No bosses on skull	Upper gums swollen; lower red
No. XIX. Mr. Thomas Smith, 1876, 'Pathological Transact.,' vol. xxvii	20 months, female, December. A former attack had appeared in April, when child was 13 months old	Both thighs and legs swollen cold and immobile (pseudo-paralysis); slight prominence in neighbourhood of trochanter; crepitus below hips and below knees. P.M.—Blood extravasation below periosteum of femora and tibiæ, and, to slight extent, fibulæ; fracture at both ends of each femur, respectively below and above the epiphysial line; no callus; blood extravasation into deeper layers of muscles	Crepitus obtainable just below shoulders; no swelling of soft parts of upper limbs	—	No stomatites
No. XX. Dr. Barlow, Case 1, 1876	4 years, male, September	Great pain in lower limbs; unable to stand. Legs rickety	—	—	Spongy
No. XXI. Dr. Barlow, Case 2, 1877	2 years, female, April	Right thigh swollen, tender, immobile (pseudo-paralysis)	—	—	Slight swelling of gums appeared after the affection of the thigh
No. XXII. Dr. Barlow, Case 3, 1878	2 years, 9 months, female, January	—	Both humeri thickened in lower third, right more than left; great tenderness, right side (pseudo-paralysis), so that forearm hung down immobile; no crepitus	—	No sponginess
No. XXIII. Dr. Barlow, Case 4, 1878	10 months, male, February	Swelling about epiphyses of both knees; knees kept semi-flexed; no fluctuation in joint	Right elbow kept flexed	—	—
No. XXIV. Dr. Barlow, Case 5, 1878	2 years, female	Left thigh swollen and tender, and strongly flexed; swelling along shaft of left femur	—	Had "bosses" on frontal	Gums extremely spongy

Skin, temperature, and general nutrition.	Heredity.	Previous health and signs of ordinary rickets.	Food and dwelling.	Progress and duration.
Anæmia; earthy complexion; extreme weakness; sleepless. Slight albuminuria. Temp. 98°. No enlargement of lymphatic glands, spleen, or liver	Second child; nothing suggestive of congenital syphilis	Both ulnæ curved; ribs beaded; chest compressed laterally; fontanelle widely open. Dentition good, 8 teeth at 10 months, and 12 teeth at 14 months. Was well till 6 months, then diarrhœa and vomiting for a short time. No diarrhœa for last 4 months	Nestlé's food at 2 months; subsequently arrowroot and isinglass on account of diarrhœa and vomiting; then Nestlé's food again. Healthy locality	Total duration not exactly stated. Marked improvement in 3 weeks, leaving slight thickening of right tibia. Was treated on raw-meat pulp, cow's milk and water thickened with potato gruel, and prepared bread.
Anæmia, cachexia, some wasting. Temperature a little elevated (had bronchitis and small patch of consolidation). A little eczema about nates and genitals for one month	No reason to suspect syphilis. Three children, all full time; this the second	Large fontanelle; ribs decidedly beaded. First tooth at 12 months. Diarrhœa since 6 months	Suckled for 3 months; then fed on cow's milk and corn flour	This attack lasted 3 months. Died probably from lung affection, but was extremely marasmic when admitted. Former attack lasted probably 3 months.
Dirty, sallow complexion. Temperature not stated, but skin was cool	—	Rickety legs and wrists	Had hysterical objection to meat; cried and vomited when offered to him	Compelled to take meat and vegetables, and within 14 days gums were all right, and he could stand, holding a chair.
No ecchymoses. Nose bleeding	—	—	Mother's milk and bread for 12 months, after that bread, beef tea, milk; no vegetables, no meat. Child had hysterical objection to meat and vegetables; would not touch them	In 14 days, after having been compelled with great difficulty to take meat and vegetables, she was improving, and in 3 days more was still improving.
—	—	Thorax characteristically rickety; lower ends of radii enlarged. Child had never walked nor talked. Much head sweating; head large	—	She was brought on account of acute swelling and tenderness of the humeri. This was of 3 days' duration, and was obviously something supervening on the old rickety condition. She was not brought again, so that the progress could not be traced.
Purpura of legs, arms, forehead, and body appeared before bone condition	—	—	—	The purpura and bone condition had appeared 2 or 3 days before he was brought, the purpura one day before the bone condition. Not brought again, so that progress could not be traced.
Cachexia	The child had cranial bosses and spleen enlargement. She was suspected to be syphilitic. History imperfect	Rickety	—	The general swelling of the thigh rapidly diminished, and left the femur thickened for a short time. Sponginess of gums rapidly disappeared. No mercury or iodide of potassium given, but only antiscorbutics. The cranial bosses and spleen enlargement persisted.

No. of case and name of observer.	Age; sex; period of year.	Lower limbs.	Upper limbs.	Skull and other bones.	Gums.
No. XXV, Dr. Barlow, Case 6, 1881. *Vide* text	8 months, female, August	Left thigh kept strongly flexed; some deep thickening felt along lower third of femur; extreme tenderness. P.M.—Not completely examined, but some blood found under periosteum, near upper end of femur, also some blood round crista ilii. Many of the muscles very pale and pulpy in parts where no actual blood clot present	Lower end of each radius much enlarged; pseudo-paralysis left wrist	P.M.—Small subperiosteal blood extravasations found on both parietals near fontanelle. Periosteum of ribs thickened, granular; much chocolate-coloured, fine flaky débris between rib and periosteum; ribs extensively bare, wasted, brittle	Small ecchymoses in gums
No. XXVI. Dr. Barlow, Case 7, 1881. *Vide* text	17 months, female, October	Both lower limbs swollen and tender. P.M.—Blood extravasation under the periosteum of both femora and both tibiæ; fracture just above the lower epiphysis of each femur and below the upper epiphysis of each tibia; no callus; blood extravasation into deeper muscular layers. *Vide* Plate	During life both scapulæ swollen in infra-spinous region. P.M.—Blood extravasation under the periosteum in infra-spinous fossa and in the venter; slight new deposit of bone from the upraised periosteum on ventral surface. *Vide* Plate	P.M. — Slight thickening near sutures — query rickety	No stomatitis
No. XXVII. Dr. Barlow, Case 8, 1882	10 months, female, May	Left leg swollen and tense; tibia and fibula thickened; upper two thirds of right leg swollen, and lower half of right femur thickened; skin pale; no local heat; much tenderness; limbs kept extended	—	—	"Have often swollen at different times;" are swollen and purplish now around the two lower incisors
No. XXVIII. Dr. Barlow, Case 9, 1882	5 months, male, August	Swelling of left femur and left tibia near junction of shaft and knee epiphysis; subsequently extension upwards, along lower third of shaft of femur. Similar swelling in neighbourhood of right knee to less extent. After subsidence of swelling there was slight displacement backwards of the shaft of left tibia below the junction with its upper epiphysis	—	—	No stomatitis

Skin, temperature, and general nutrition.	Heredity.	Previous health and signs of ordinary rickets.	Food and dwelling.	Progress and duration.
Ecchymoses both upper lids; proptosis (slight) one eye. Temperature 99·8. Extreme cachexia. P.M.—Some localised tubercle in left lung found, with blood-stained serum in pleura and ecchymoses on parietal pleura	No reason to suspect syphilis. Mother delicate, other children rickety, but not showing any sign of syphilis	Never strong; head-sweatings since 3 months; bowels at one time constipated. Two months ago severe diarrhœa, after which legs became tender. Wrists large; no teeth at 10 months	No breast milk; condensed milk first, then cow's milk, then various foods; Anglo-Swiss food when bone-symptoms appeared. Abode healthy	Total duration 2½ months. Had been taking cod-liver oil for 2 months when brought. Was ordered raw-meat juice. Seemed a little better for a week only; gradually sank. Fatal.
Temperature elevated, but this from measles; skin had been cool previously; cachexia; general tenderness	Fifth child. Child was not regarded as syphilitic, but there was the history of the first child being born dead, and of this child having snuffled occasionally as a baby, but had no rash. The fourth child suffered from laryngismus and fits	Beading of ribs and characteristic rickety signs found post-mortem in humeri and radii, which did not show hæmorrhagic features; head-sweating excessive; laryngismus from 9 months old. A weakly child since 4 months. Much diarrhœa at 15 months till onset of acute bone symptoms	Breast fed entirely for 7 months, and partly till 11 months old; subsequently, and at time when bone-symptoms appeared, beef tea, bread, puddings, a little cow's milk; no vegetables. Abode said to be healthy	Duration 2½ months. Was ordered, when admitted, raw-beef juice, mashed potatoes, and orange juice, but there was no time for this to act, as the child was then in the incubation stage of measles, of which she died in less than a fortnight.
Skin cool; emaciation; anæmia. No diarrhœa or vomiting before, but they occurred during the illness	Father had had chancre twenty years before. Four other children said to be healthy. This child had rash over the trunk when one month old, but no snuffles	Wasted since 1 month; no diarrhœa or vomiting; ribs slight beading; no enlargement of lower ends of radii	Breast for 4 months, afterwards Ridge's food, with water only	Had attended three or four times before swelling of leg appeared. When the swelling appeared she was ordered meat juice and orange juice, which she took greedily. Within the next week there was slight extension of swelling, but the week after it had began to subside rapidly. The sponginess of gums had also gone. The child ceased to attend, but died within 2 months. Cause obscure.
Not generally hot. Temp. once 99·6° (ten days after onset)	No reason to suspect syphilis: The mother has had her children rapidly	Frequent offensive diarrhœa; was brought on account of tetany, laryngismus, and fits; ribs markedly beaded, and all epiphyses large	No breast milk; two pints of cow's milk in 24 hours, with half the quantity of water, thickened with a little Neave's food	Was brought on account of tetany, and then it was noticed that the left knee was strongly flexed. The swelling was noticed ten days after this, then he was ordered lemon juice and meat juice and wet compresses. For about a week there was slight extension of swelling up the left thigh, and at one place on the outer side, just above epiphysis, slight fluctuation became perceptible. Compresses and antiscorbutics were continued. Swelling subsided in a week. Slight thickening of both femora and left tibia remained for a fortnight, then everything cleared up with exception of slight displacement of shaft of left tibia below junction with epiphysis; this much less marked when the boy was seen, 3 months later, having still large rickety epiphyses. The duration of the illness may be considered 7 to 8 weeks. Bowels much improved when on antiscorbutic diet.

No. of case and name of observer.	Age; sex; period of year.	Lower limbs.	Upper limbs.	Skull and other bones.	Gums.
No. XXIX. Dr. Barlow, Case 10, 1882. *Vide* text	15 months, male, November	Left thigh and leg swollen, tender, flexed; right leg a little swollen. After subsidence of swelling left femur and tibia thickened and right tibia slightly thickened	Both wrists enlarged, right considerably so; swelling extends for about an inch upwards from epiphisis	Slight thickening of frontal in front of fontanelle; extremely weak in the back	Not swollen
No. XXX. Dr. Barlow, Case 11, 1880	14 months, male, February	Left leg swollen and tender from knee to ankle; a little swelling above the right ankle-joint.	—	—	Upper gums spongy and sw len; lower gu a little swolle putrid sm Swelling of gu preceded limb affection
No. XXXI. Mr. Shoppee's Case, 1881	11 months, male, August	Both thighs and legs swollen, tender, extended, immobile (pseudo-paralysis); swelling especially of middle of shaft of each femur	Wrists became enlarged one month after thighs	Back very weak	Spongy and tremely swolle bled freel odour putr The spongin of the gums peared after onset of limb affection

Skin, temperature, and general nutrition.	Heredity.	Previous health and signs of ordinary rickets.	Food and dwelling.	Progress and duration.
No local heat of limbs; rectal temp. 101° five weeks after onset, but three days after this 99·4°, and subsequently normal; extreme anæmia; earthy complexion; general tenderness	Father healthy, mother small and poorly developed, but with no particular ailment. Child was born before term; said to have snuffled occasionally	Head-sweating since 3 months old; first tooth at 12 months; bones noticed to be thick near joints for several months; ribs beaded; slightly knock-kneed on right side	Breast milk for 6 weeks, then no fresh food for 14 months; groats and Swiss milk, then baked flour, then Nestlé's food, then Robb's biscuits, then Liebig's extract, finally Swiss milk and saccharated lime-water. Abode healthy	Bone-symptoms, &c., had lasted 5 weeks, when he was put on anti-scorbutic food, and cold compresses were applied. Rapid subsidence of swelling, and tenderness. Some thickening of left femur remained for several weeks, but within eight weeks was able to stand with a little support. Salt douches and shampooing were employed after the first month, and Ol. Morrh. ʒij, daily after first week.
Skin cool; anæmia; sallow complexion; great fretfulness; bowels once or twice daily; stools not specially offensive	No reason to suspect syphilis. This the second child; the first child said to be healthy	Two teeth at 6 months, has eight now; thorax scarcely at all rickety. Was pretty well till last 3 months	No breast milk; Swiss milk at first, Ridge's food when 2 months old; lately oatmeal and beef-tea; no vegetables	Gums had been bad for 3 months in spite of various treatment. Was ordered lemon juice, raw meat, cow's milk, and cod-liver oil, and to be brought again if he did not improve. Was not brought again.
Extreme anæmia; emaciation; general tenderness; conjunctival hæmorrhage. For a short time hæmaturia and uric acid in the urine, which was scanty (this at the onset and only for a short time)	No syphilis; no hæmophilia. Two other children had been subject to hæmaturia, associated with passing uric acid crystals	Fairly nourished till 8 months, when he had bad diarrhœa and slight attack of measles; diarrhœa again at 10 months, followed by present illness. Two teeth at 8 months; large epiphyses	Nestlé's food almost entirely. Abode healthy	Total duration 3 months. Limb condition had begun to subside a little, and the tenderness diminished, when the child got bronchitis, of which he died.

DESCRIPTION OF PLATE VII.

Cases described as "Acute Rickets," probably a Combination of Scurvy and Rickets (Dr. THOMAS BARLOW).

FIG. 1. Scapula: subperiosteal hæmorrhage and new-bone formation in upraised periosteum.

FIG. 2. Scapula: vertical section, showing subperiosteal hæmorrhage on the venter and on the infra-spinous fossa.

FIG. 3. Tibia: subperiosteal hæmorrhage and fracture just below the junction of shaft and epiphysis.

FIG. 4. Femur: subperiosteal hæmorrhage; fracture just above junction of shaft and lower epiphysis.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

E. Burgess, & J. H. Morgan, del. Wardour. Brook Chromo Lith.

SUBPERIOSTEAL HÆMORRHAGE

(PROBABLY SCORBUTIC)

OF

THREE LONG BONES IN A RICKETY INFANT.

BY

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Received January 29th—Read March 27th, 1883.

ON October 20th, 1882, a male infant, æt. 9 months, the first child of a young mother, was sent to me by Dr. Taylor, of Willesden, under whose care it had been for a week, on account of great swelling of the left thigh and leg. The child was extremely wasted, pale, and ill, and was obviously in much pain. The mother gave the history that until four weeks ago the child was thriving, when a lump was noticed below the left knee. This lump had gradually grown larger and more extended, and the thigh also had become affected. There was no history of injury.

On examination the shafts of the tibia and femur were found to be, or gave the impression of being, enormously enlarged. The limb was excessively tender and motionless, but there was no redness, and the knee- and ankle-

joints were not involved. There was a slight amount of œdema, and the superficial veins were unduly prominent. I failed to detect any certain fluctuation. For the last week a swelling had been noticed below the right knee also, and the upper third of the right tibia was found considerably swollen and tender, but without fluctuation. For the last few days a swelling had been seen at the right wrist, and there I found a typical rickety enlargement of the lower end of the radius. A like enlargement had been observed by Dr. Taylor at the lower end of the right tibia. The ribs were markedly though not extremely beaded. No other bones were in any way enlarged. Inquiring further into the history it was learned that the child immediately after birth was fed on Swiss milk, and that at the age of three weeks it was given Nestlé's milk food, on which and on Savory's food it had been reared up to this time, milk—Swiss milk—having been given in only very small quantities.

Looking to the condition of the wrist and the ribs, it seemed to me that the enlargement of the two tibiæ and of the femur must be in some way or other connected with rickets, but the absence of fluctuation rendered the nature of the swelling most obscure, and new growth appeared out of the question. An entire change in the mode of feeding was advised, but the child really seemed too ill to promise any hope of recovery. No history or suspicion even of syphilis could be elicited, but mercurial inunction was nevertheless suggested in addition to iron and cod-liver oil, treatment which had been already begun by Dr. Taylor.

On the following day the child came again, having, unknown to Dr. Taylor, been brought to see Dr. Samuel West, and with Dr. West I then again saw it. The parents were extremely anxious that something more definite should be done, and agreeing with Dr. West in his opinion that at one point in the thigh, though at one point only, there was deep-seated fluctuation, and that no harm could happen therefrom, a trocar and cannula were

passed at that place. The instrument went down to bare bone, and there escaped a few drops of sanguineous fluid. The necessity of incision through the periosteum at once became obvious. The child was sent home, and having followed it in the evening, I made, with the help of Dr. Taylor, who administered chloroform, free incisions in both thigh and leg. Femur and tibia were alike found to be entirely denuded of periosteum throughout their whole shafts, and lying between the bones and their covering were huge blood-clots, which had to be broken up with the finger before any part of them could be removed. Hardly any fluid escaped, and the wounds showed no undue tendency to bleed. Drainage-tubes were inserted, and hot fomentations were ordered for the left limb, and also for the swelling on the right tibia, which it was deemed well to leave unopened, at any rate for a day or two, in the hope that it might subside.

The subsequent history may be told in a few words. The child at once began to improve, and the swelling of the right tibia gradually disappeared. The broken-down blood-clots were expelled by the help of syringing, and on November 13th the drainage-tubes were finally removed. Except from the immediate track of the wounds, which were perfectly closed early in December, there was never any purulent discharge.

I saw the child again on January 16th, 1883, and found it to all appearances thriving, playful, and well. The shafts of the affected bones were then of normal size, the periosteum had obviously resumed its natural position, and the only indications of the former mishief were the scars, with slight thickening of the soft parts beneath them, and inability to bring the limb to perfectly full extension. This latter defect, however, was rapidly diminishing. The beading of the ribs had gone, and the right radius, although still different from the left, was much smaller than it was. The teeth were being cut properly and the child was beginning to stand. There is no hæmorrhagic diathesis in the family.

Although, with the complete history before us, it may now seem strange, yet it is nevertheless a fact, that the difficulties of diagnosis were in this case very considerable. Had there been anything like distinct fluctuation of the swollen leg, it is obvious that diagnosis would have been easier and more certain; but in the absence thereof, due doubtless to the fact that blood-clot was tightly packed between periosteum and bone, no nearer diagnosis could be hazarded than that the condition was in some way or other connected with rickets. I am inclined now to think that the disease was rather scorbutic than rickety. The rearing of the child could not well have been worse, deprived as it had been of all fresh food, of that kind of food, in fact, the want of which so unquestionably induces scurvy.

If we seek to explain the hæmorrhage under the periosteum as the consequence of a rickety periostitis, we are met at once by the objection that periostitis is extremely uncommon even in the worst cases of rickets, and although it may appear extraordinary that the thickened and highly vascular state of the periosteum in severe rickets does not more frequently pass these pathological limits and involve the membrane in true inflammation, the facts are otherwise, and after the lapse of two and twenty years are still in harmony with the teaching of Sir William Jenner in his classical lectures then delivered on rickets, wherein he says: "Of Meyer's opinion that rickets is an inflammatory affection of the periosteum and endosteum I shall only say that my many examinations of rickety children after death have enabled me to lend no support to such a notion; that I have seen no sign of pre-existing inflammation of the bone or its covering, although I have carefully looked for such" ('Medical Times and Gazette,' vol. i, 1860, p. 466).

And again he writes, in a passage which bears especially on this case: "The periosteum of the whole bone is often more vascular and thicker than natural, but in the many post-mortem examinations of extreme rickets which I have

made, I have never seen any bloody fluid as described by Guérin beneath that membrane."

The striking point, however, in this case was the mass of clotted blood surrounding the shafts of two long bones, and probably also part of the shaft of another; but looking to the fact that the detached periosteum retained its vitality and became once more adherent to the bones, which themselves also lived, I cannot help thinking that inflammation of the periosteum was a less essential element of the pathological change than the extravasated blood. In the early stages of acute periostitis from injury, we sometimes find a blood-stained fluid between the periosteum and the bone to be rapidly followed by purulent discharge and death of the affected shaft. Here, however, there was blood-clot, the pus was very small in quantity, and all the parts have lived. Be it noted also that, although the periosteum was separated from the entire shafts, the neighbouring joints were unaffected and the cutaneous surface showed no sign of inflammation. True, the extravasation of blood, the local blood-letting, so to say, may have been the salvation of the periosteum acutely inflamed, and have preserved it from a destruction which otherwise it might not have escaped; but may not a better explanation of the case be found in the fact that there had long been at work a cause such as that which leads to blood-extravasation in purpura and scurvy, diseases induced by lack of proper nutrition, and characterised by copious hæmorrhages and extravasations in different parts of the body.

Do other cases lend support to this doctrine? I have been able to find only one recorded case of the kind, and that was brought before the Pathological Society by Mr. Thomas Smith in 1875, and was entitled "Hæmorrhagic Periostitis of the Shafts of several of the Long Bones, with Separation of the Epiphyses" ('Transac. Patholog. Soc.,' vol. xxvii, p. 219).

The patient was a girl, æt. 23 months. She had been suckled up to three months of age, and beyond a slight

attack of diarrhœa had had fairly good health. There was not the least evidence of syphilitic taint, and there was but slight rickety enlargement of the ends of the ribs. The child could stand and walk alone. Eleven months before admission there were swelling and tenderness of both lower limbs commencing in the feet. This disappeared until two months before admission when the feet again began to swell. When admitted the child lay on its back with its legs stretched out and motionless. The whole of each lower limb was swollen. The hip- and knee-joints were unaffected. The temperature ranged between 99° and 101·5° F., and death took place suddenly six days after admission.

On examination the following lesions were found symmetrical as to the two sides. Complete separation of the epiphyses of the femur, and a large amount of recent blood-clot between the periosteum and the shaft, which was smooth and white. No suppuration was to be found anywhere in connection with the bones. The structures in the hip- and knee-joints were healthy. Blood was found uniformly effused through the deeper layers of many muscles of the thighs. In the leg, besides effusion into the muscles, there was similar detachment of the periosteum and separation of epiphyses from the tibia and fibula, with a large amount of extravasated blood around the denuded shafts. Crepitation could be easily felt at the shoulders, but the upper limbs were not examined. There was no disease of the abdominal viscera, but in the middle of the lower lobe of the left lung was a wedge-shaped patch of congestion as large as a chestnut, with an abrupt edge, of a reddish-purple colour. There was no history pointing to a hæmorrhagic diathesis. From the report of the discussion upon this case ('Lancet,' vol. i, 1876, p. 14) we learn that "Mr. Smith had excluded syphilis, rickets, purpura, &c., as causal conditions, and thought the hæmorrhage was from the diseased and inflamed periosteum."

The numerous extravasations, however, which were found in other parts in Mr. Smith's case, appear to me to

lend strong support to the theory of the scorbutic origin of the subperiosteal hæmorrhages, and to the suggestion which I have ventured to make as to the nature of the hæmorrhages in my own. The suggestion, too, gains further point from a case which, with two others, formed the text of a most instructive clinical lecture by my colleague, Dr. Cheadle, "On Three Cases of Scurvy Supervening on Rickets in Young Children" ('Lancet,' vol. ii, 1878, p. 685).

In all three cases the most prominent symptoms of scurvy had been superadded to those of rickets, and in the second of them "some hard swellings could be felt deeply seated in the flesh of each thigh, *and the shafts of the long bones felt enlarged and swollen.*" Can there be much doubt that the enlargement of the shafts of the long bones was in that case also due to subperiosteal hæmorrhage? The other and more ordinary signs of scurvy there clearly told what unhealthy and unnatural agents had been at work, and a certain knowledge now of the physical cause of the like swollen and enlarged shafts in my own case throws light on the obscurity which in all probability involved that case no less than this. The three children referred to by Dr. Cheadle had "been fed on a *scurvy* diet, which comprised neither milk, nor fresh vegetables, nor fresh meat;" and it was upon a "*scurvy* diet" that this patient, whose history I have related, had been reared for the first nine months of its life. The rarity and interest of the pathological condition, and of the history throughout, would be my excuse for bringing an isolated case before the Society, had I not the hope that its record and consideration may help towards a surer diagnosis and better treatment in other cases like it, where the absence of swollen and bleeding gums deprives us of an all-important clue to the nature of the malady with which we have to deal.

CAVERNOUS NÆVUS OF THE RECTUM

PROVING FATAL IN AN ADULT

FROM HÆMORRHAGE.

BY

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Received February 13th—Read April 10th, 1883.

The following case, as a pathological and clinical rarity, appears to deserve some special notice.

R. K—, æt. 45, was sent to me as an out-patient at University College Hospital on March 13th, 1882, by the kindness of Dr. Sayer, of George Street. The patient was at this time a fine-looking, very tall, and muscular man, most cheerful, and in fact a type of rude health. He complained that eight months ago he suffered from diarrhœa for four or five days with considerable loss of blood at each stool. This was stopped by a single dose of medicine, since which he has been much troubled by constipation, rarely having a motion without medicine, and being sometimes a week without relief. At intervals "something like a bladder seems to burst" in his rectum, and blood with mucus escapes. A motion on the 13th

was described as "tape-like" about half an inch broad. He has suffered no pain at any time but has frequent desire to go to stool (sometimes every half hour) with much straining. He denies ever having had any form of venereal disease. Since boyhood he has been often obliged to strain with his motions when constipated, and at such times has passed blood. Sometimes he did not notice this for three or four years, and as a rule his bowels have been regular. On examining the rectum with a long vaginal speculum and strong reflected light, I could not discover any particular narrowing of the gut. The whole wall of the bowel was then carefully examined from the anus upwards, as far as the speculum admitted, when I found three shallow ulcers on the mucous membrane (Pl. VIII, D) described below in the post-mortem notes. They were seated on some smooth longitudinal folds in the wall of the gut, of a yellowish colour and suggesting a quantity of fat in the submucous tissue. The ulcers though shallow exuded continuously a considerable amount of blood. Their base, however, presented a peculiar mottling of a purplish colour, as also did the surface of the irregular folds alluded to, the whole picture giving rise to the suspicion that we had to deal with a nævoid mass in the wall of the bowel. The statement of the patient, too, that similar bleedings had occurred on and off since boyhood seemed to lend support to this view. There was none of that anxious look about the man which one observes in those in whom cancer of the rectum has advanced to the bleeding stage. Reviewing the whole question for the students present, I expressed the opinion that the patient was probably suffering from a nævoid lipoma of the rectum, some of the most superficial parts of which had become abraded and ulcerated. He was, therefore, ordered to keep quiet and to use an injection of equal parts of Tr. Fer. Perchlor. and water, morning and evening. This treatment produced no improvement; he still went to stool twelve to fourteen times daily with much bleeding, and at the end of the week as the man

was becoming feeble and anæmic, I admitted him into hospital on April 19th with a view to more radical examination and treatment. He then came under the care of Mr. Marshall, who has kindly permitted me to record the subsequent notes of the case. His abdominal and thoracic organs were now very carefully examined, but nothing abnormal was found. He stated, however, that a few days before admission he had passed a formed motion "as thick as three fingers and six inches long," confirming the view that there was no stricture. On April 20th Mr. Marshall examined the patient under ether and with the aid of a vaginal speculum and No. 7 rectal bougie. He expressed the opinion that the disease was probably ulceration of the sigmoid flexure of the colon. He ordered enemata of extract of logwood night and morning with sulphate of copper and opium internally, due attention being paid to the bowels.

Every day after this the patient passed considerable quantities of blood, but his stools were much less frequent than when up and about his work. He became rapidly weaker, and on May 2nd his appearance is noted as showing all the evidences of extreme anæmia; he also felt very weak and giddy on getting up, and even when in bed blood oozed from the anus. Ergotine was then given night and morning in three-minim doses, but without any effect. On May the 4th he was seized with great general tremor followed by the loss of a large quantity of blood from the rectum, which appeared to flow almost continuously from the anus throughout the following night. On the 5th he became very restless and could hardly be restrained though quite conscious. In the afternoon he was quieter, but after raising himself in bed he fell back and died. A particularly disagreeable odour was perceived from the body immediately after death.

The notes of the post-mortem examination, as recorded succinctly by Mr. Stanley Boyd (then our registrar), may perhaps be given in full with advantage in a rare case of this kind.

"May 6th, 1882 (post-mortem twenty-three hours after death).—Temperature of room 62°. Rigor mortis well marked. Body splendidly developed and well nourished, not quite cold. In right pleura twelve ounces of deep orange-coloured clear fluid, in the left eleven ounces, but no signs of inflammation on either side. Pericardium universally adherent by old but not very tough adhesions. Heart: considerable clot on right side extending into great veins and pulmonary artery; scarcely decolorised; valves on right side healthy; mitral valves thickened but quite competent; lunulæ of aortic valves much fenestrated, contiguous portions of two adherent for about one third of an inch from their attachments, held together by a firm nodule of inflammatory tissue; endocardium of right auricle considerably stained; heart substance is pale and quite yellow in patches, consistence lessened, but little fat on surface. What blood there was about the body was coagulated. Left lung normal on surface, excessively œdematous, consistence nowhere diminished; right as left, but tissue of lower lobe is commencing to soften. Peritoneum: nothing abnormal, no fluid. Liver not projecting below right ribs, upper surface irregular; section shows lobules marked out much too clearly, they are very plainly seen everywhere; consistence everywhere increased but mostly so at anterior border of right lobe. Spleen: size normal, section pale, tolerably firm. Kidneys: left pale, normal; right normal; suprarenal capsules small, apparently too soft, section normal. Stomach normal, and contains curdled milk. Cæcum and descending colon full of fæces, the rest almost empty until the rectum is reached, then more fæces are met with. Mucous membrane of large gut normal throughout, no blood seen anywhere (Pl. VIII, A.). Wall of rectum in lower four and a half inches is much thickened by a nævoid growth in its walls which gives a purple colour to the mucous membrane (B. to C.). There are three or four prominent longitudinal folds here, each three quarters of an inch or more in width. The two largest are on the left side of the bowel; these are

the folds felt during life. One of them, just to the left of the middle line in front, and one and three quarters and two and a quarter inches from the margin of the anus are two ulcers (Pl. VIII, D) one not as large as a threepenny piece, the other and higher having an irregular shape and measuring one and a half by three quarter inches, a thin slough of mucous membrane adhering to it, while the smaller ulcer leaves the submucous tissue bare. Brain normal."

It is hardly necessary to say anything more about the tumour except that it presents everywhere the characters of cavernous nævoid tissue. This can be well seen in the specimen which I have brought down to the Society (see Pl. VIII, B. to C.) and in some sections under the microscope which were prepared by Mr. Stanley Boyd (see

woodcut). Such a tissue in this situation must be of extremely rare occurrence, for after careful search I have failed to find any recorded instance in which it has been noticed. Fatal bleeding from such a growth must be still more rare.

* One half of the above specimen is in University College Museum, No. 5795, and the other half in the museum of the Royal College of Surgeons (Path. Catalogue, Series xxiv, No. 2578).

DESCRIPTION OF PLATE VIII.

(Cavernous Nævus of the Rectum, A. E. J. Barker, F.R.C.S.).

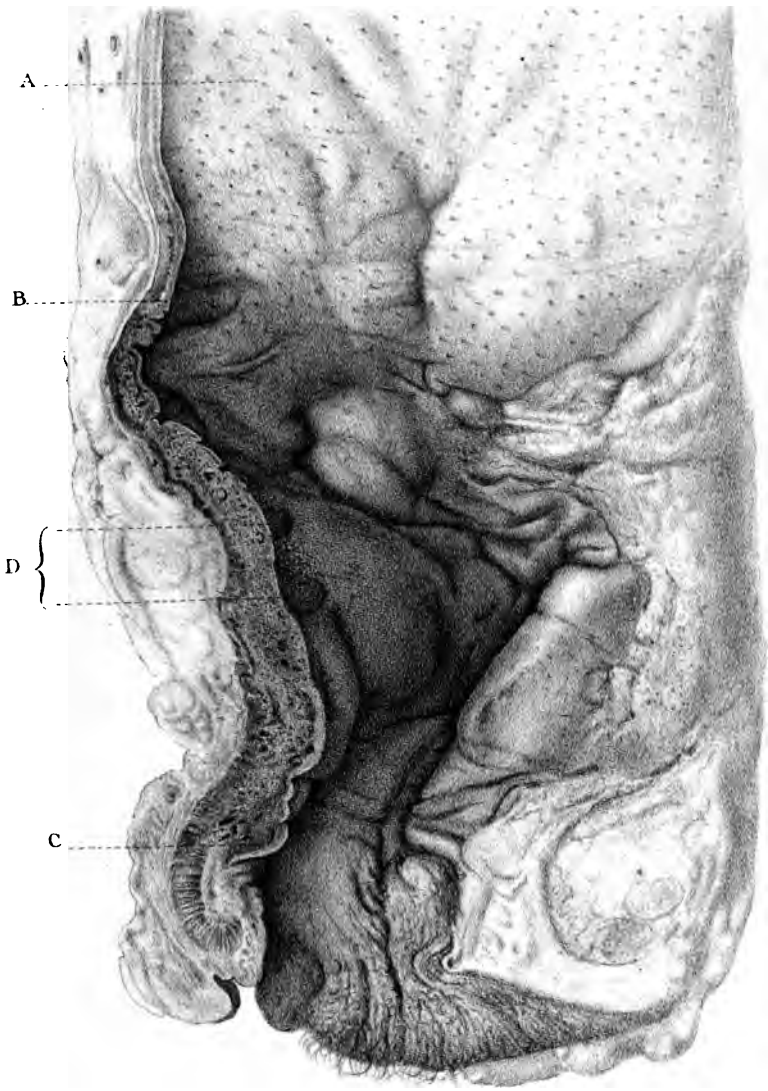
This specimen shows about nine inches of the rectum with the anus, preserved in spirit (half size).

A. Normal surface of bowel.

B to C. Extent of cavernous nævoid change.

D. Half of two ulcers from which the fatal bleeding took place.

From B to C. The typical cavernous tissue is seen in section.


A
B
D
C

I.

A CASE OF PURULENT PERICARDITIS TREATED BY PARACENTESIS AND BY FREE INCISION,

WITH RECOVERY.

II.

THE STATISTICS OF PARACENTESIS PERICARDII

(WITH REMARKS).

BY

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(Received February 21st—Read April 24th, 1883.)

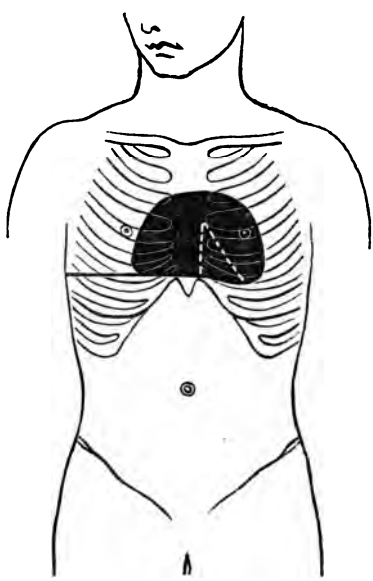
I. A Case of Purulent Pericarditis treated by Paracentesis and by Free Incision with Recovery.

Alfred N—, æt. 16, a van boy on the railway, was admitted into the Hospital for Diseases of the Chest, Victoria Park, on September 7th, 1882, when the following history was obtained. Two months previously to his illness he had been struck in the back by a passing truck and knocked down. He felt a little pain in the right side for a week afterwards, though he did not trace any connection between the accident and his present illness. He had been in apparently robust health, and in active work until August 16th, when he was attacked with shivering and with pain in the left side and front of the chest, and his breath became so short that he was obliged to take to his bed. The pain was relieved by strapping the side firmly, and in a few days he was able to get up. On September 4th, three

weeks later, he went out for a short walk, but became suddenly very faint and almost fell down. Severe pain seized him "in the pit of the stomach," and his breath became shorter than ever. The symptoms gradually increased until September 7th, when he came to the hospital with great dyspnœa, deeply cyanised and almost pulseless, and was admitted at once.

On the night of admission he was frequently sick, was much distressed by dyspnœa, and hardly slept at all.

On the next day (September 8th) his condition was as follows: The patient was a well-nourished, strong-looking lad. He lay upon his back. He was deeply cyanosed. The respirations were 28 and laboured. The pulse was 78 at the wrist, markedly paradox, *i.e.* intermitting with each inspiration, and losing thus two or three beats with each intermission. The heart was beating regularly at about 120 a minute. The præcordial region was bulging and slightly œdematous, and the epigastrium looked full. The apex beat was not visible, nor could it be felt. There was an indistinct heaving felt over the whole præcordium, and impulses could be faintly felt in the third, fourth, and fifth spaces midway between



the left nipple and the sternum. Dulness extended in the fifth intercostal space from close to the right nipple line to nearly three

inches outside the left nipple line. The area of dulness was somewhat conical in shape and reached upwards to the middle of the second intercostal space near the sternum. (See diagram.)

The cardiac sounds were almost entirely inaudible except just where impulse was faintly felt. All round the limits of this area of dulness fine crackling crepitation (pleuritic) was heard, which extended also into the left axilla, but the lungs elsewhere were not affected and the bases behind were perfectly natural.

The liver dulness, which commenced at the sixth rib in the right nipple line, was increased for nearly an inch downwards and was distinctly felt below its normal position in the epigastrium. There was slight œdema of the feet, and also of the front part of the chest, chiefly over the area of dulness. The urine was acid, of 1030 specific gravity, and contained no albumen. There was no swelling of any joint.

The diagnosis was made of acute pericardial effusion, probably, it was thought, of rheumatic origin, for it transpired that the patient had suffered from flying joint pains a few days previously. The patient was ordered to have twelve leeches to the præcordium, and the bleeding to be encouraged with poultices.

The subsequent daily notes are as follows:

September 9th.—The leeches took well, and the bites bled freely during the night. The patient was sick during the night, and took but little food. He had some snatches of sleep. This morning he complains of no pain, but of great weakness, and of a nasty taste in the mouth. The patient is less cyanosed. The respirations are 32. the pulse 72, paradox, and feebler than yesterday. The area of præcordial dulness is slightly less than it was yesterday, and the heart sounds appear louder and audible over a wider area than they were. There is no trace of friction sound.

10th.—Patient slept fairly, and the sickness has stopped after some effervescing mixture with strychnia. He seems easier and has taken more food. Resp. 32, pulse 80, of the same characters as yesterday.

11th.—Patient slept fairly. Feet a little more swollen. Area of dulness not further diminished, but the heart sounds are louder. Crepitation as before round edges of dulness. Pulse fuller, still intermitting, but feeble beats occasionally felt during intermission, at the wrist 78. The heart beating regularly 104 (showing a loss of 26 at the wrist). Resp. 40.

12th.—Slept fairly. Is more cyanosed to-day. The dulness is again a little increased, and the heart sounds are feebler. Pulse 80 (paradox), resp. 28.

13th.—Patient slept badly, and had a cold sweat last night. He is more cyanosed. Pulse at wrist 60, paradox, and feebler. Heart beats 104 and regular. Resp. 32. The area of dulness has increased since yesterday; the whole præcordium is more prominent. There is an indistinct sense of impulse outside the left nipple. In the epigastrium immediately below the sternum is a slight depression, and from the difference in resistance the liver probably is pushed down.

14th.—Slept fairly during the night, though a little disturbed by sickness. The cyanosis is greater, and there is more œdema of the feet. Pulse 64, very soft, and easily compressible, and paradox. Resp. 32. The heart beating regularly at the rate of 120. The præcordial dulness reaches to the right nipple. I had made up my mind that paracentesis pericardii was called for, but before deciding finally I invited Dr. Hensley to see the patient with me. He agreed in the diagnosis and in the advisability of tapping. A puncture was then made with a small trocar and cannula connected with a syringe lent me for the purpose by Dr. Hensley. The place chosen was the fourth intercostal space, almost immediately below the left nipple. The cannula was two inches long and was cautiously pushed in up to the hilt in a direction straight backwards. Pus was obtained in the syringe, and, as it was very viscid and ran slowly, the tube was connected with an aspirator, and in this way thirteen ounces of laudable sweet pus were removed. Three ounces of 1 per cent. carbolic acid solution at a temperature of 100° F. were injected twice into the cavity and allowed to flow out again, bringing with them about one ounce more pus, thus making the quantity removed about fourteen ounces in all. The patient was very greatly relieved, became much less cyanotic and "smiled." The lines of dulness receded after the operation about one inch in all directions, and did not reach now above the upper border of the third rib. The pulse, which before the operation was distinctly paradox as hitherto, became much less irregular, some inspirations producing no intermission. Beyond some shrinking and a little pain on the first prick of the needle, no distress was occasioned to the patient by the operation. The depression in the epigastrium, where the feeling of resistance was diminished, became less distinct, and the liver appeared to have risen somewhat higher after the operation. Microscopically the pus teemed with rod-shaped bacteria. It was simple pus and contained no shreds or flocculi. 9 p.m.—The patient has had a little pain in the seat of puncture, but was otherwise more comfortable. He says that he feels warmer. Pulse 80, less intermittent; resp. 30. Morph. Hydrochlor. gr. $\frac{1}{12}$th by mouth.

15th.—Slept well. Patient says that he feels quite different from

yesterday; he feels hungry and has taken food well. Except a little pricking at the seat of puncture he has had no pain anywhere. Patient is still very cyanotic. Dulness is slightly diminished, pulsation is felt now in third and fourth interspaces to left of sternum, and here the heart sounds are loud over an area of about two inches. No friction or murmur. Pulse 80 at wrist, still paradox. Heart 104, regular. Urine 1008, acid, no albumen. A good deal of fine crackling crepitation (pleuritic friction) all round the area of dulness, and over it on the left side nearly as far as the sternum.

16th.—The patient slept fairly after one twelfth of a grain of morphia by mouth. The appetite is fair. Patient is rather more cyanosed than yesterday. There is no decrease of the area of dulness, but the præcordium seems less bulging. Pulse 64, intermitting every three beats, rather small and feeble. Heart sounds 112, showing a loss of 58 at the wrist, that is, of two for each inspiration, the respiration being 28. 9 p.m.—The patient is not feeling so well; he complains of a sense of pain and weight in epigastrium, and there is more dyspnœa and cyanosis. Pulse 72.

17th.—Patient slept fairly after one sixteenth of morphia, but he feels worse this morning. He still has pain in epigastrium, and has been sick a few times. The dyspnœa and cyanosis is greater. There is an increase in the area of dulness, and the heart sounds are feebler. Pulse 70, intermittent; heart 115, regular; resp. 33. 1 p.m.—Patient was found to be very blue, and much distressed, complaining of very great pain, referred to the middle of the abdomen. The cardiac dulness seemed as large as at first. The condition of the patient was one of such urgent danger that I decided to perform paracentesis again. The puncture was made in the fifth interspace, in the left nipple line, with a trocar and cannula measuring about $\frac{3}{16}''$ in diameter. A slight preliminary incision in the skin was made with the scalpel. This was followed by an attack of retching which lasted for a few minutes. A trocar was then passed easily and without much pain into an enormous cavity, the end of which could not be reached with the whole length of the cannula (about $4\frac{1}{2}''$). The pus came away with great difficulty although the aspirator was used. Only about four ounces were obtained, and on pushing a probe down the cannula backwards and forwards, several ounces more escaped with difficulty. The pus was extremely viscid, flaky and slightly offensive. With the view of aiding the flow, about four ounces of 1 per cent. carbolic-acid solution were slowly injected. This passed in fairly easily; it caused a good deal of pain and did not assist the flow at all, for it was only after long probing that some few ounces more of pus could be

evacuated. It was then decided to make a free opening. Chloroform was carefully administered by Mr. Bateman, and as soon as slight anæsthesia was obtained, a narrow-bladed, sharp-pointed bistoury was thrust along the side of the cannula, which I used as a director, into the cavity. This was followed by a copious discharge of pus, which was ejected on expiration a distance of four or five inches from the chest walls. When the cavity had fairly emptied itself, the opening was enlarged by means of the probe-pointed bistoury and a dressing forceps, care being taken to make the internal opening into the sac free. Air entered into the cavity on inspiration, and air and pus escaped on expiration. The dulness became very much less at once, but how great the diminution was could not be clearly made out on account of the entrance of air. It is difficult to say how much pus escaped but it must have been a very large quantity, for the whole bedding was soaked; it was estimated at at least two quarts.

The patient recovered much of his colour immediately after the operation and was greatly relieved. The pulse ceased to be distinctly paradox, and became almost regular. The patient took chloroform well, and without any alarming symptoms. Mr. Bateman and Mr. Hind assisted in the operation. The cavity was freely washed out with a warm solution (1 per cent.) of carbolic acid, and a large drainage-tube ½″ in diameter inserted for about four inches. About two ounces of 1 in 40 solution of carbolic acid were injected before dressing and left in the cavity, and the wound was covered with picked oakum and carbolised cotton wool. The patient was transferred to another bed and soon fell asleep.

At 9 p.m. the dressings and draw-sheet were quite saturated with discharge and were changed. Beyond a little pain in the left side the patient feels "all right." Pulse 108, not intermitting, slightly dicrotous. Resp. 48, chiefly abdominal. Cyanosis much less.

18th.—Patient slept fairly after one twelfth of a grain of morphia. He feels weak, but better. He has taken food well. The tongue is clean, and the temperature not raised. The cyanosis is much less. Pulse 108, much stronger, and no longer intermittent; resp. 34. 5 p.m.—Patient says he is quite comfortable. His face looks a little flushed, but the colour is red rather than blue. Respiration quiet, 34. Patient complains no longer of shortness of breath. Pulse 120, quite regular both in force and frequency, and of good volume. There has been no sickness since the operation. The præcordial area is now everywhere resonant from the air in cavity. No *bruit de moulin* or friction sound, and the heart sounds clear. The dressings are soaked with pus, which is quite sweet. The drainage-tube was removed, and a soft French catheter (No. 21) inserted; a little pain

was caused by its passage between the ribs. About eight ounces of a solution of carbolic acid (1 in 40) were injected by means of a syphon. It did not return easily through the catheter, which was then removed, and the pus and solution allowed to escape of themselves. After well washing the cavity out, as the opening seemed inclined to close too rapidly, a piece of the resistant catheter was inserted and tied in, the dressings of picked oakum and carbolised cotton wool being applied as before. While being dressed the patient had an attack of faintness, which passed off, however, on administering a little brandy. At 10 p.m.: patient had been a little light-headed during early part of evening, but had slept fairly. Resp. 40, pulse 120, regular.

19th.—Patient slept fairly after morphia (grain one twelfth). Pulse 120, slightly irregular in force, regular in time; resp. 32. Chest dressed, the tube removed, and about two ounces of very thick curdy pus, with a slightly fetid odour and of a brownish colour, came out. The catheter was replaced. Up to the present there has been no escape of anything but ordinary pus and the usual curdy flakes. 3 p.m.—Patient was put slightly under chloroform, and after removing the tube I inserted the little finger of my right hand into the opening; the finger passed in for its whole length. The line of the incision had become, from the contraction of the walls of the cavity, almost horizontal, running towards the sternum. The tip of the finger only just reached the cavity, and the heart was then felt beating upon it. The cavity was freely syringed out with carbolic-acid solution, which brought away a few ounces of pus containing some curdy flakes. A flexible india-rubber drainage-tube, ¼″ in diameter, was inserted. At 9 p.m. patient had pain in bowels, probably from constipation. Pulse 112, regular; resp. 24.

20th.—Patient has slept well; the appetite is better; the pain in abdomen was relieved by an action of the bowels after an enema. Pulse 108, regular; resp. 32. The chest was dressed. A fair amount of discharge had come out during night. No discharge escaped on removing tube except a little blood, chiefly from the granulations round the incision. The tube was replaced and some carbolic acid (one in forty) left in cavity. At 9 p.m. patient seems weaker, much prostrated, and had been sick once. Pulse 120, regular; resp. 28.

21st.—Fair night after morphia (one sixth grain). Much better. Resp. 20. Chest dressed. A few ounces only of discharge in dressings, quite sweet. Cavity washed out with (one in forty) carbolic-acid solution through the tube, which was not removed. In

the evening the patient had considerable pain in stomach, which was relieved by an enema and ol. Ricini.

22nd.—Patient slept fairly after morphia (one twelfth). Pulse 112, scanty, irregular. He looks a little blue still. At 2.30 p.m. chest dressed. Opening free, discharge runs freely from tube, and is quite sweet, wool soaked with some ounces of pus. Cavity washed out with one in eighty carbolic-acid solution. The cavity seemed to admit only about one ounce at each injection. No flakes came away. Patient has a much better colour and is much stronger. Pulse 120, fairly regular, occasionally intermitting with inspiration, but only now and then. Dulness much reduced, the apex being within the nipple and the right border extending only about three quarters of an inch beyond the edge of the sternum. Respiration good everywhere, still a little dry crackling round limits of dulness. The signs of air in the cavity have disappeared, cardiac sounds are heard well and are quite natural. At no time have any unusual pericardial or cardiac sounds been audible.

23rd.—Patient had a good night without morphia. He eat a little minced meat yesterday. He is much stronger, and was found to-day reading a book. The chest was dressed. A good deal of pus had drained into dressings during the twenty-four hours. The cavity was washed out through the tube which was not removed. Pulse 98, occasionally intermitting; resp. 28.

24th.—Patient slept well without morphia. The chest was dressed. Several ounces of pus had escaped in the twenty-four hours. The tube was not removed, but the cavity was washed out as usual. The whole body was found covered this morning with a small papular urticaria-like eruption, slightly itching, some of the spots fusing on the trunk.

25th.—Rash the same, only more profuse, and still itching; it is punctate, erythematous, raised in places in the form of circular patches with a yellowish smooth centre (? Lichen urticatus). The chest was dressed, the tube removed, and the cavity washed out, the fluid returned almost unchanged. The cavity does not hold more than an ounce. The cardiac sounds are normal; nothing abnormal audible except the usual pleuritic friction. The base of the lungs perfectly healthy, as they have been throughout.

26th.—The rash is fading on trunk, though still the same upon the legs and arms. It was very irritable yesterday. The cavity was washed out as usual and the dressings changed.

27th.—Hardly any discharge in dressings. Cavity closing. The patient was allowed to-day to sit up in bed to have some solid food.

28th.—The chest was dressed. Discharge in dressings hardly

more than one ounce in the twenty-four hours. The area of dulness reached just to the right edge of the sternum, and was slightly increased upwards and outwards towards the left. A good deal of pleuritic crackling was heard all round this area. Breath sounds were audible over the whole præcordium, but were quite natural over the lungs, back and front. A probe was passed in along the track of the tube. This ran almost horizontally from without inwards, towards the base of the xiphoid cartilage. The orifice had now approached nearer the sternum, so as to be within the left nipple line and almost in the position where the apex beat is normally found. The probe impinged at once upon the heart, the walls of which did not appear rough. On directing the probe towards the xiphoid cartilage it passed apparently under the heart, the movements of which were felt distinctly along the probe all the time. The probe passed in as far as the drainage-tube went and then stopped. It could not be passed in any other direction except along this track, and the cavity seemed entirely to have disappeared except along the fistulous track which was occupied by the tube. A smaller and shorter tube was inserted.

29th.—Patient better. Pulse 100. Chest dressed, hardly any discharge.

30th.—Chest dressed as usual. Pulse 88, a little irregular.

October 1st.—Chest dressed, hardly any discharge.

2nd.—Chest dressed, about one ounce of discharge in the dressing. Pulse 108, a little irregular.

6th.—Tube changed; a smaller one, one inch shorter put in. Pulse 116, slightly irregular.

10th.—Hardly any discharge. Patient's colour almost natural. Œdema of feet and of chest wall almost entirely gone. Pulse intermits only now and then with inspiration.

13th.—Tube shortened.

14th.—Tube removed. Incision kept open by a small piece of gutta-percha tissue.

17th.—Incision closed.

20th.—During the last week the patient has steadily gained strength. The cardiac dulness is now hardly increased beyond the normal, except along fourth rib, about one and a half inches from

the sternum, and in this part impulse is very distinctly felt. The heart sounds are everywhere normal. The breathing sounds healthy, and the crepitation over the margins of the dulness is almost gone. Pulse regular. Patient moves with ease and vigour, and has no dyspnœa.

21st.—Was up for one hour, and bore it without distress, but found "his legs very weak." The heart sounds are forcible, as the result, doubtless, of the extra exertion of being up.

31st.—Patient has been up every day for the last ten days, and grows daily stronger. He has no pain or dyspnœa, but he is not allowed to move about at all.

December 12th.—Patient is up nearly all day, and feels quite well. The cardiac dulness is somewhat increased in all directions, reaching a little to the right of the sternum, the apex being in the nipple line in the fifth space.

January 2nd.—Patient has complained lately of a little pain in the seat of the scar.

February 20th.—Cardiac dulness as above, but the apex is a little farther out than it was. The only change in the heart sounds is that the pulmonary is slightly accentuated. Pulse regular and of good volume. Patient looks the picture of health. The pain that he has at times seems to be dyspeptic.

23rd.—Patient left hospital perfectly well.

At the beginning of April he stated that he had returned to his usual work, which he could do as well as ever. He had no pain or dyspnœa on exertion and felt perfectly well. The cardiac dulness was almost normal, though slightly increased upwards; heart's apex-beat in normal position.

The patient was last seen in September, and was perfectly well, following his ordinary occupation.

The case may be briefly summarised thus:—A healthy lad of sixteen was suddenly seized with dyspnœa and with pain in the front of his chest. The dyspnœa gradually, in the course of three weeks, became so severe that he was admitted, as an urgent case, into the hospital.

On admission he was deeply cyanosed, with considerable dyspnœa, pulsus paradoxus, slight œdema of the feet and of the chest walls over the præcordium, and with all the ordinary physical signs of a large pericardial effusion. The temperature being not raised, it was assumed that the effusion was of some standing, and probably rheumatic in

origin. No permanent improvement occurred in spite of copious local bleeding by leeches, and on the sixth day after admission the symptoms became so urgent that paracentesis pericardii was performed, with the result of removing about fourteen ounces of pus. The patient was greatly relieved, the pulse became less paradox, and the physical signs of effusion diminished; three days later the symptoms again became urgent, the effusion had largely increased, and a second paracentesis was performed with the removal of about sixteen ounces of pus.

As the pus was slightly fetid it was then decided to make a free incision into the pericardial sac. This was done, using the cannula as a director. A very large amount of pus escaped, and air entered the sac. The operation was not antiseptic, except that all instruments were washed in 1-40 carbolic acid solution, and the cavity syringed out with a solution of the same strength. A drainage tube was inserted, and the wound dressed with oakum and carbolised cotton wool.

The pus contained, beyond numerous rod-shaped bacteria, nothing except what would come from an ordinary abscess cavity, namely, shreds of lymph and lumps of curdy pus.

The patient was at once relieved, the pulse became regular, and the cyanosis much less. The cavity rapidly contracted, and the discharge diminished. Two days later, under chloroform, the finger was inserted into the sac, and the heart felt beating upon the tip of it above.

A week after the operation the patient had an attack of general urticaria, which lasted for three days. From this time the patient made a rapid and uninterrupted recovery. The cavity contracted, so that at the end of twenty-seven days the tube was removed, and on the thirtieth day the incision had completely closed. The heart sounds were natural throughout. No murmur or pericardial friction developed. There was no affection of the lungs or pleura beyond some dry pleurisy immediately round the sac. The pulsus paradoxus disappeared, as well as the dyspnœa and the slight œdema of the feet and chest walls.

At the end of five weeks after the operation the patient was able to sit up, and from that time rapidly gathered strength. The cardiac dulness for a short time remained almost normal, within the limits to which it had slowly returned, but as the patient began to move about and exert himself, which he was soon able to do without any discomfort, the cardiac dulness slowly increased in size, and on examination, eight weeks after the operation, it extended about half an inch to the right of the sternum, the apex being just outside the nipple line. The patient complained that, if he exerted himself, he felt a little pain occasionally in the left side, near the seat of the scar. As he gathered strength this increase slowly subsided, and two months later the cardiac dulness had returned almost to its normal limits. These alterations in the cardiac dulness are, it appears to me, strongly confirmatory of the diagnosis. The lad grew much in height, and increased considerably in weight during his stay in the hospital.

In the absence of the confirmatory evidence of a post-mortem examination, it will be right in a case of such rarity as the present to state the grounds upon which the diagnosis of pus in the pericardium was made.

There was evidence of a large effusion of fluid in the anterior and lower part of the thorax. The area of dulness corresponded in position, extent, and shape with those usual in pericardial effusions. There was the usual absence of cardiac impulse, and the heart sounds were extremely weak. There was evidence of great obstruction to the circulation: first, in the enlarged liver, and in the œdema of the feet and chest walls; secondly, in the dyspnœa and cyanosis; and, lastly, in the character of the pulse (pulsus paradoxus). The peculiar, elastic, semi-fluctuating depression in the epigastrium which developed just before the last paracentesis I regard as additional evidence of the effusion having its seat in the pericardium.

The diagnosis was confirmed by the subsequent history of the case:

1. By the great and immediate relief given by the operation of paracentesis and by the subsequent incision; a relief which is, as will be shown later, the especial characteristic of the removal of even small amounts of fluid from the pericardium.

2. By the evidence given by the trocar in the cavity, and by the finger inserted through the incision, for the heart was felt beating upon the end of the trocar and upon the tip of the finger.

3. By the variations in the cardiac dulness, which were observed during the patient's convalescence.

Two physical signs only are wanting to complete the case clinically, viz. the *bruit de moulin* and pericardial friction.

The *bruit de moulin*, or mill-wheel sound, which is met with where air and fluid exist together in the pericardium, is an extremely rare phenomenon. When it occurs it is no doubt pathognomonic of pyo-, or hydro-, pneumo-pericardium, but the converse is probably not true, that it always occurs under these conditions, for the analogous phenomenon in the pleura, viz. succussion, is not invariably present in pyo- or hydro-pneumothorax.

The absence of pericardial friction is a greater difficulty, for at no time throughout the illness was undoubted pericardial friction detected. Once only the heart sounds were described as sticky, as they frequently appear to be in rheumatic pericarditis, just before or just after the usual to-and-fro friction. Yet the single fact of the absence of friction is not sufficient to outweigh the concurrent testimony of the other physical signs, for cases of undoubted pericarditis are recorded in which pericardial friction has been absent throughout; and it is stated, although only to be denied, by Bauer in 'Ziemmsen's Encyclopædia,' that in purulent pericarditis friction is usually absent. A case will be quoted later (Case No. 76) which clinically was in most respects similar to this; the

pericardial effusion was also purulent, and there was throughout no friction.

To the absence of these two physical signs, viz. the *bruit de moulin* and pericardial friction, too much importance, then, must not be attached, for the former is of doubtful value, and the absence of the second admits of explanation.

Clear, however, as the case is clinically, it becomes still clearer, when we consider what other diagnoses were possible. The possible pathological conditions appear to be these:

1. An abscess making its way upwards from below the diaphragm.
2. A localised empyema.
3. A suppurating cyst, or an abscess, in the anterior mediastinum.

The first supposition, that of an abscess making its way up from below the diaphragm, from the liver, or from between the liver and diaphragm, may be set aside at once, for there is no evidence to support such a diagnosis.

If it were a collection of pus in the anterior mediastinum, we must suppose that it developed immediately in front of the pericardium, in such a way that it pushed both the pericardium and the heart neither up nor down nor outwards, but bodily backwards away from the anterior walls of the chest. This is an almost impossible pathological assumption.

Such a collection of pus, not in the pericardium, would be due either to a localised empyema, or to an abscess or suppurating cyst in the mediastinum. In either case there would certainly have been some dulness posteriorly in consequence of the displacement of the heart, and this there was not. The circumscribed area of dulness corresponding so exactly with that of pericardial effusion would be very remarkable, and it would be difficult to explain the cardiac symptoms.

Of empyema there was at no time any other physical

signs, and it is difficult to conceive how a cyst or abscess in the mediastinal tissue could have developed in this situation, in the direction of greatest and not of least resistance. It is, I think, almost impossible that a mediastinal cyst should have been limited to this situation.

The course of the case, the rapid contraction and closing of the cavity after removal of the fluid, make it clear that the pus was confined in a very resistent and elastic sac, and there are only two conditions which would provide such a sac, viz. a thick-walled mediastinal cyst (of the existence of which there was no other evidence) and an effusion into the pericardium.

I may quote in this connection a very remarkable case, in which the diagnosis was made during life of a mediastinal cyst, and in which paracentesis was several times performed with great relief, but which proved to be on post-mortem examination a case not of mediastinal cyst, but of chronic pericardial effusion. This case was under the care of Sir Risdon Bennett at the Chest Hospital, Victoria Park, to whom I am indebted for permission to refer to the clinical history of the case.

The patient, Charles E—, was a youth, æt. 20. He was admitted with symptoms of mediastinal pressure, and the diagnosis was made at first of mediastinal tumour, though it was subsequently altered to that of a cyst in the mediastinum. Paracentesis was performed, and a large quantity of clear serous fluid evacuated, which on cooling coagulated and deposited a large amount of cholesterine. The heart, which could not be localised, was found immediately after the tapping in its proper place. The patient was tapped several times, but only on the first occasion did the fluid contain cholesterine. Each time the tapping gave great relief. He was in the hospital four times altogether, for some months on each occasion, and on the last occasion for one year and three months. At this time the patient had considerable anasarca, and a large amount of fluid in the peritoneum. He died gradually of exhaustion four years and a half from the commencement

of the illness, and four years from the date of his first paracentesis. The post-mortem showed that what had been regarded as a mediastinal cyst was really a pericardial effusion.

The pericardium was greatly distended, extending from some distance beyond the right edge of the sternum to more than two inches outside the left nipple line. Its cavity was filled with slightly blood-stained serum. The whole parietal portion of the pericardium was enormously thickened, not only just under the sternum, but also behind and at the root of the lungs. Two inches outside the left nipple the pleura and pericardium together measured more than one inch in thickness, the internal surface was smooth and had not lost its polish, except where one or two long adhesions stretched from it across to the heart, which lay in the upper and right portion of the sac, some inches away from the apex. The heart was small and covered entirely by a soft, velvety, villous layer of recent lymph which easily peeled off. In the new membrane were numerous gray tubercles. The left pleural cavity was obliterated. There were a few gray tubercles in the liver and spleen, but none in the kidneys. The peritoneal cavity was crossed in all directions by dense adhesion, and contained a large quantity of turbid serum. The intestines were matted together and covered with recent lymph, in which as well as in the parietal layer of the peritoneum were scattered numerous gray tubercles. The liver was adherent to the abdominal walls and the capsule much thickened. It weighed 3 lb. 11 oz., was cirrhotic and granular, the fibrotic change being most marked at the periphery beneath the capsule.

I am unable to give the full clinical notes of this remarkable case, for it has not yet been published, but I quote it in connection with this paper as an instance of what had been during life always supposed to be a mediastinal cyst, proving to be on post-mortem examination a large pericardial effusion, for which paracentesis was several times performed with great relief to the patient. The case is of geat importance in reference to that at present under consideration, for it lends strong support to the diagnosis.

After all that has been said, the conclusion is clear that we have to do here with a purulent pericardial effusion, for

with this diagnosis the clinical facts agree, while the assumption of any other diagnosis creates far more difficulties than it solves.

Purulent pericarditis is rare, and generally asssociated with some disease of itself incurable, for example, pyæmia, tubercle, or with some eruptive fever, &c. Primary pericarditis of any kind is uncommon, and primary purulent pericarditis, of which the present case is an instance, may be placed among the rarest of clinical rarities.

Though the evidence of pericardial effusion was conclusive, there was nothing in this case to indicate that it was purulent. The temperature was never raised, and even after the operation only once reached 100° F. There were no rigors, nor sweating, nor did the patient present any symptom which would suggest suppuration except the œdema of the chest walls, which was most marked over the præcordial region. The value, however, of this sign was discounted by the existence of œdema of the feet, a very common clinical phenomenon in cases of pericardial exudation, especially in young persons.

The operation in itself was extremely simple. More courage was required to perform the first paracentesis than to make a free incision with the knowledge that the effusion was purulent. Chloroform was administered with the view of avoiding the shock of the operation, and to keep the patient still while the knife was being used. There was, of course, some risk in giving chloroform, but no other anæsthetic was available at the time, and I thought the risk of even chloroform less than that which I should run by operating without any anæsthetic at all. The patient took chloroform well.

The operation being urgent, free use of antiseptics could not be made, although I had determined, if time had been given, to have had the operation performed under the spray with the usual antiseptic precautions. The result, however, could under no circumstances have been more satisfactory, for the patient recovered without a

single bad symptom at any time, the discharge and wound remaining throughout perfectly healthy.

The spot chosen for puncture was fixed as far to the left as possible, because in ordinary cases of paracentesis the heart is at this place farthest away from the chest walls. The objection to tapping so far out is that the trocar would puncture the pleura before reaching the pericardium, and on this account a spot is usually selected nearer the sternum, within the point to which the pleura usually reaches. In this case I felt safe in operating farther away from the sternum because I had reason to believe, from the duration of the disease, and from the signs of dry pleurisy, that the pleura was adherent over the præcordium, and that I should therefore run hardly any risk of opening the pleural cavity. The result proved the correctness of this supposition.

The fluid had, however, no sooner been evacuated than the cavity contracted and the incision, which had been direct, became horizontal, so that a long sinus formed. I did not quite expect this, and it would be an argument for adopting in the future a spot for puncture nearer to the sternum.

Of the large amount of fluid which escaped, and of the great relief immediately given to the patient, I shall speak again in discussing the cases recorded in the appendix to this paper.

The epigastric depression which developed just before the incision is a physical sign which has been described in some instances of pericardial effusion, but is a rare phenomenon. It is, I believe, due to bagging of the pericardium and central portion of the diaphragm, and may have indicated that the abscess would have burst, if it had not been opened, through the diaphragm into the peritoneum.[1]

The quantity of urine before and after the operation

[1] Since writing the above a similar observation has been recorded in the 'Lancet' of January 27th, 1882, in a case of pericardial effusion, under Dr. Clifford Allbutt's care.

was not observed. In some cases which have been described recently, stress is laid upon the great increase noticed in the amount after paracentesis. The decrease is, I believe, simply due to the congestion of the kidneys, and the increase to the relief of their congestion by the operation.

The urticaria which developed over the whole body a few days after the operation is a curious clinical phenomenon, but one which has been observed commonly in cases of tapping of hydatids of the liver, and occasionally also after paracentesis of the pleura and peritoneum.

The *pulsus paradoxus*, or *pulsus cum respiratione intermittens*, which was so exquisite in this case, of which a tracing is given, is a very rare phenomenon. Pulses which vary with respiration are not at all uncommon in the minor degrees, especially in cases of heart disease; but the regularity is then nearly always cardiac in origin, that is to say, the irregularity of pulse is due to irregularity of the heart's action. The true pulsus paradoxus is essentially different. Here the heart continues to beat regularly, though the pulse intermits, and this intermission occurs during each inspiration, lasting as long as inspiration lasts. In the present case the heart throughout continued to beat regularly at the rate of about 112 in the minute. The pulse, however, varied. In some instances the difference between the heart and pulse beats were almost exactly twice the number of respirations, *e.g.* heart 112, pulse 64, respiration 28. $64+56=120$. After the operation the pulse became perfectly regular.

Nearly all the instances recorded of pulsus paradoxus have occurred in connection either with pericarditis or with affections of the mediastinum. Griesinger records a case in which the cause was found, post mortem, in a constriction of the aorta by bands of fibrous tissue surrounding the vessel. Kussmaul records other cases, and adopts this explanation, and seems to regard the pulsus pardoxus as pathognomonic of a condition which he names mediastinitis fibrosa. The mechanism in these cases appears to

be this: fibrous bands form round the great vessels, which are attached to the sternum and ribs in front. As the sternum and ribs move forwards on respiration the bands are tightened and the aorta constricted. In most of the cases, however, besides the mediastinitis there was also pericarditis, either with adhesion or with effusion, and Traube and Bäumler record cases of pulsus paradoxus with simple pericardial effusion, and without any mediastinitis, and one instance is on record where it was observed in a case of very large left pleuritic effusion.

The simplest explanation is no doubt that of Kussmaul, if only the pathological condition it assumes were constant, but this is not the case. Two theories have been suggested for the pulsus paradoxus of pericarditis. The one, like Kussmaul's, is anatomical, and refers the interference to stretching of the pericardium, where it is reflected round the vessels by the descent of the diaphragm. The other is rather physiological, and refers the interference to the feeble condition of the heart, which has only just sufficient power to produce the pulse at the wrist during expiration, the slightly increased resistance offered to the exit of blood during inspiration proving too much for its strength. This I cannot regard as an adequate explanation, for if the heart was so feeble as the theory presupposes there would be more clinical evidence of cardiac weakness in these cases, and the pulsus paradoxus would be a more common phenomenon.

The only experiments I know conducted with the view of determining the mechanism of the pulsus paradoxus are those mentioned recently by Prof. Rosenbach.[1] An air ball was introduced into the right pleura and so adjusted that it compressed the inferior vena cava as it passed through the diaphragm. The pulsus paradoxus was produced in its most typical form. The author concluded therefore that another possible explanation of the phenomenon lies in the insufficient filling of the heart with blood, owing to the interference with the circulation

[1] Art. "Pericarditis," in Eulenburg's 'Real-Encyclopädie.'

through the inferior vena cava by constriction of its mouth, as it passes through the diaphragm.

This is a most important addition to our knowledge of the pulsus paradoxus, and will probably explain those cases in which it has been observed with simple pericardial effusion, or with the displacement of the heart in large pleural effusions. It is remarkable that the only reference to the pulsus paradoxus which I find among the cases of paracentesis pericardii which I have collected is in that of Prof. Rosenstein's, which in this as in every other respect is the counterpart of the present case. To this case I will now shortly refer:

A boy, æt. 10, was brought to Professor Rosenstein's clinic with the history of fourteen days' illness, during which time he had had pain in the epigastric region, dyspnœa, a dry cough, and some fever. He was found to have a large pericardial effusion. As the temperature was hardly raised above the normal and there was no œdema, surprise was felt when, on inserting a fine syringe, pus was drawn off. Paracentesis was at once performed in the fourth space on the left side, near the edge of the sternum, and 620 cc. of laudable pus removed. The pulse which before the operation was 140 and intermittent (paradox), became regular and 92. The respiration dropped from 52 to 28. A few days later the patient was found to have fluid in the left pleura, which was also tapped and 1100 cc. of serum removed, and at the same time 120 cc. more of pus were removed from the pericardium. The patient, however, was in a critical condition, there was great dyspnœa and cyanosis, the veins of the neck were much distended, œdema of the feet and scrotum had appeared, and the pulse was extremely paradox. The temperature, however, throughout had never risen above 100·5° F. A free incision was now made into the pericardium in the fourth left space near the edge of the sternum and a large amount of pus escaped. Two drainage-tubes were inserted. The patient was wonderfully relieved at once. A week later the pulse was regular and the œdema gone, and in three weeks after the operation the wound had completely healed. A little later the signs of pleuritic effusion reappeared on the left side, and paracentesis was again performed with the removal of 1000 cc. of pus. A day or two later a free incision was made with the evacuation of about 1500 cc. of pus. In six weeks' time the wound in the pleura also had completely healed and the patient was discharged perfectly well, having gained considerably in weight, fifteen weeks

from the commencement of his illness and ten weeks from the date of the incision of the pericardium.

The case of Prof. Rosenstein's just referred to and that of which I have given an account in the present paper are the only cases recorded in which purulent pericarditis has been treated like empyema, by free incision.[1] It is remarkable that in both recovery was rapid and complete, and with the exception of those rare instances in which inspissated or caseous pus has been found unexpectedly in the pericardium post mortem, and which have been regarded as evidence of the possibility of spontaneous cure, these are the only cases of purulent pericarditis in which recovery has taken place.

II. The Statistics of Paracentsis Pericardii, with remarks.

Paracentesis pericardii is essentially an operation of the present century. It was advocated, it is true, by Senac in 1749, and it had been suggested exactly 100 years before by Riolan, but from that time up to the early part of this century no practical advance had been made. Those writers who referred to the operation contented themselves with either advocating or opposing it on purely theoretical grounds.

Van Swieten went so far as to describe a method, but never carried it out. Corvisart spoke of the operation in 1806 as too heroic to be justifiable. Laennec, however, describes it as practicable, though he records no cases in which it was performed. It was not until 1819 that paracentesis pericardii was for the first time performed, and that too with success, by Romero, of Barcelona, who operated upon three cases, two out of which

[1] At the end of the table, p. 281, will be found appended a brief note of a recent case which has occurred since the reading of this paper.

which recovered. Prior to this the operation is stated to have been performed in 1798 by Dessault, and 1810 by Larrey, but Dessault's case was an encysted pleurisy, and Larrey's is not conclusive, so that Romero's claims to priority are, I think, established. Eight years later Jowett, of Nottingham, punctured the pericardium with a trocar, the fluid escaping into the pleura, from which it was absorbed.

No other operation is recorded until the year 1839, when Schuh tapped the pericardium at the request of Prof. Skoda in a case which proved ultimately to be one of mediastinal tumour. Two punctures were made, but without result. In the next two years a most extraordinary series of cases was published in Russia during an outbreak of scurvy. They were remarkable not only for their success, but also for the nature (blood) and for the large quantity of the fluid which was removed.

In 1854 Trousseau wrote his memorable essay upon the subject, recording cases of his own and of M. Aran. This attracted great attention in France, and consequently we find a series of cases in French literature in the course of the next few years.

Dr. Clifford Allbutt was the first to introduce the operation into this country, which was performed upon his advice with complete success by Dr. Wheelhouse in 1866, and again by Dr. Teale in 1869.

Since the year 1865 the operation has been frequently performed in France, Germany, and England, and a few cases have been recorded from America. Of the seventy-nine cases recorded, fifty-three are subsequent to the year 1864.

In 1881 a further great advance was made by Prof. Rosenstein, of Leiden, who after twice tapping the pericardium in a case of purulent pericarditis, finally laid the sac freely open under antiseptics and inserted a drainage tube. The case was treated exactly as if it had been one of empyema. The result was entirely satisfactory, for the boy recovered completely. The present case is the

second of this kind recorded, and the first in this country.

I have collected all the cases of paracentesis pericardii which I have been able to find recorded, and I have added to them not a few others which have not yet been published. The difficulties of collection were much increased by the frequent double reference to the same case, in the literature of the subject, under the names both of the physician and of surgeon concerned. I have endeavoured as far as possible to identify these cases, and where they have been quoted under two names I have bracketed these names together. The references given I have been at pains so far as possible to check.

The cases number seventy-nine; I have arranged them in chronological order, and have given in successive columns the sex, age, clinical history, and result. I have made a short note of the method and place of operation, of the nature and amount of the fluid removed, and in fatal cases I have added, when possible, a brief abstract of the pathological conditions found. I propose to discuss the subject to a great extent according to these headings.

Sex.—Of the seventy-nine cases the sex was specified in seventy-one, and of these fifty-seven were males and fourteen females. For this remarkable prevalence of males I can find no explanation.

Age.—In twelve the age was not stated. The rest I have arranged in periods.

	Under 10	Up to 15	-20	-25	-30	-40	-50	-60	-70	Total.
Males . . .	6	6	8	13	10	6	1	1	2	53
Females . . .	1	4	2	2	3	1	...	...	1	14
Totals . .	7	10	10	15	13	7	1	1	3	67

The distribution is fairly equal in the different periods

of early life. From the facts as regards age as well as sex, we may conclude that the majority of the cases was probably not of rheumatic nature. Otherwise there would be a greater proportion among young persons. This further agrees with the clinical experience that, as a rule, rheumatic cases recover without the necessity of operative interference.

The causes.—The accompanying table shows the diseases with which the pericarditis was associated:

Disease.	Number of cases.	Recovery.	Death.
Phthisis	13	4	9
Purulent pericarditis	12	2	10
Rheumatic fever	11	7	4
Scorbutus	9	6	3
Pleurisy	6	5	1
Injury	3	2	1
Pneumonia	2	...	2
General dropsy, due to *a.* Morbus cordis	2	...	2
General dropsy, due to *b.* Nephritis	2	2	...
General dropsy, due to *c.* Chronic bronchitis	1	1	...
Mediastinal tumour	1	...	1
Unassigned	17	7	10
	79	36	43

Rheumatic fever.—Of the seventy-nine cases, in twelve only did the pericarditis occur in the course of rheumatic fever, and one even of these is doubtful. Of the remaining eleven, seven recovered completely, and of the four fatal cases two were relieved by the operation, and one of them ought almost to be called a recovery, for the patient lived for six months after the operation. The other lived for six days. Of the remaining two, in one the operation was performed so late that the patient was moribund at the time, and in the last the operation was in itself fatal, the trocar penetrating the right ventricle, and the patient dying of hæmorrhage into the pericardium in a few minutes. This, it may be stated, is the only instance among these cases of a fatal result from the operation itself.

The amount removed was small, a few ounces only, except in one case, where it was as much as two pints.

The doubtful case was one of purulent pericarditis, and though there was a history of joint pains, the disease was probably not rheumatic fever, but rather some form of pyæmia, for purulent pericarditis is very rare in rheumatic fever, and joint pains resembling rheumatism are not uncommon in the course of diseases of pyæmic or septicæmic nature.

In six of these cases the attack was acute, and in four it was the first attack. In the remaining five the disease was chronic. It is worthy of note how great was the relief given by the operation in all the cases.

The analysis of this table confirms the preceding conclusion that the great majority of the pericardial cases, which were severe enough to require operation, were not cases of rheumatic origin.

Scorbutus.—Nine cases are recorded of paracentesis pericardii in the course of scorbutus, all occurring in males. They were remarkable, first, for the nature, and secondly, for the large amount of fluid which was removed. It was in all cases hæmorrhagic, and varied from one and a half to five pints. Six recovered completely. One died six hours after the operation, and the other two at later periods. Two cases, which lived six months and six years respectively, were found on post-mortem examination to have the pericardium adherent.

Phthisis.—It has long been remarked that chronic pericarditis like chronic pleurisy is commonly associated with tubercular disease, and to a certain extent this is borne out by these cases, for we find no less than thirteen of them to have been cases of phthisis; and to this number may be added six cases of pleuritic effusion and four cases of phthisis which on post-mortem examination proved to be associated with tubercular disease, making altogether twenty-three cases (or nearly thirty per cent.) in which the lungs and pleura, one or both, were affected.

Of the thirteen cases of phthisis seven had pleuritic

effusion as well, so that we seem to trace a connection between phthisis, and pleuritic and pericardial effusion.

All the phthisis cases occurred in the course of chronic illness. Four were so far relieved by the operation as to be put down as recoveries. Two lived for more than two months, and one for nearly one month. Only two died within a short time of the operation (eight hours and two days). In none could the death be attributed to the operation itself but rather to the pre-existing disease.

The effusion removed was in all cases serous, and except in four cases the quantity was but a few ounces. The larger quantities were 22, 27, 28, 35, and 49 ounces.

Pleurisy.—Of the six pleuritic cases the effusion into the pericardium was purulent in two, one of these being a case of empyema. In the others it was serous. One only of all these six recovered. Most were relieved by the operation, though one died in a few hours and another in six days.

General dropsy.—In five cases the pericardial effusion occurred in the course of general dropsy, as the result in two of morbus cordis (both of these patients died), in two of nephritis (both of these recovered), and in one of general bronchitis (this case also recovered).

Pneumonia.—In the two cases of pneumonia the effusion was purulent. They were both probably of pyæmic nature, and will be referred to subsequently with the purulent pericarditis cases.

Miscellaneous.—In three cases the pericarditis followed exposure to cold, a surgical operation, and a mediastinal tumour respectively, and in another case it was referred to injury.

Unassigned.—Of the remaining seventeen cases, in which the cause is not assigned, six recovered and eleven died. Post-mortem examination was made in six, and the pericarditis was found in four cases to be due to tubercular disease, associated in two cases with tubercle in both lungs and pleura, and in another with tubercle as well in the peritoneum. In one of the remaining cases double

hydrothorax was found, and in the other a pericardial effusion with adherent pleura.

Purulent pericarditis.—I have reserved to the last an account of this perhaps the most interesting group, viz. purulent pericarditis. Twelve cases are recorded, all fatal with the exception of the two last, those of Professor Rosenstein and myself, which were treated by free incision. In only one case did death take place within a few hours of the paracentesis, some of the cases surviving to have the operation repeated many times, in one case as often as six times.

The quantity removed varied a good deal, but the average was higher than with serous effusion, and in two cases the amount is described as considerable.

The cause of the affection is by no means clear. Six of the cases were probably pyæmic in origin, though this is not quite certain. Two were associated with empyema, although in one the empyema was subsequent to the operation.

The relation of tubercle to purulent pericarditis does not appear to be established by these returns, for among the cases of tubercular pericarditis recorded in the table, the effusion was serous in several, but in none was it purulent or hæmorrhagic.

All the cases were in males, and seven out of the ten in which the age is given were between ten and twenty years of age, the oldest of all being only thirty-one.

It is worthy of notice that in no case was the diagnosis of the nature of the effusion made previous to the operation, nor does there seem to have been any physical sign by which the diagnosis could have been determined.

Length of illness before operation.—The illness in the great majority of cases had been of long duration, in several of some months. The longest on record is that of Sir Risdon Bennett, in which the effusion was present for more than four years. In only twenty-five cases (or 1—3) was the duration short, and the attack therefore more or less acute.

The effect of the operation.—The immediate result was in nearly every case good. It is remarkable how great was the relief given even by the removal of only a few ounces of fluid, in some cases this appearing to be sufficient to lead to the complete absorption of the rest.

One case only is recorded in which the operation itself was fatal, and this was due to laceration by the trocar of the right ventricle. Six cases died within twenty-four hours of the operation, but with these deaths the operation itself had nothing to do. The one case, which died within two hours of paracentesis was moribund at the time of operation. Twelve persons lived for a week after the operation, eight for about a month, six for a period longer than one month but less than ninety days, three lived for a little more than six months, and twelve lived very much longer, and in some others recovery appears to have been complete.

When the mechanical effects of the effusion were obviated by paracentesis, death it appears was the result not of the pericarditis or the paracentesis, but of the disease to which the pericarditis was due.

The nature of the fluid.—In ten cases the fluid was pure blood; nine of these occurred in scorbutics, the tenth was a case of hæmorrhage into the pericardium in consequence of laceration of the right ventricle. In twelve it was purulent, and of these only those two recovered which were treated by free incision. In the rest the effusion, where it is specified, was serous.

The quantity of the fluid removed.

Up to 5 oz.	-10	-15	-20	-30	-40	-50	Above 50
13	16	9	8	12	11	4	11
46				38			

The amount is tabulated in the table. Any quantity

above one pint may be regarded as large, and it will be seen that where less than one pint was removed in forty-six instances, more than one pint was removed in thirty-eight instances.

The largest quantity occurred in the hæmorrhagic (scorbutic) cases: 3½, 4½, 5, and 5½ pints; but even serum has been met with in large quantities, and as much as 2 and 2½ pints have been removed by operation. On post-mortem examination, in a case recorded by Sibson, serum was found to the amount of 3½ lbs., and in another case,[1] the amount measured three litres, *i.e.* rather more than five pints, while Kyber records a still more remarkable case, in which the pericardium was filled with blood to the incredible amount of 10 lbs.

The number of punctures.—In many cases the operation was frequently repeated. In sixteen cases the pericardium was tapped twice, in three thrice, in one six times, in one eight times, in one many times over a period of four and a half years; and in two, after twice puncturing, the pericardium was incised and laid freely open.

The mode of operation.—The methods employed have been various. The earliest operations, those of Romero, were performed by making an incision through the skin and muscles in the intercostal space over the pericardium, exposing the pericardium, picking it up with a pair of forceps, and opening it by knife or scissors, the fluid being allowed to flow away.

It is noteworthy that with this, according to our present views coarse, way of operating, the results were satisfactory.

Trousseau incised the skin and exposed the pericardium and then punctured with a trocar. In one case he passed a gum elastic sound through the opening into the sac.

Lobel used a large injecting syringe with a needle which he removed each time after filling the barrel of the syringe, and re-inserted.

Since the invention of the aspirator by Dieulafoy, this

[1] 'Recueil de Mem. de Med. Militaire,' 1880, 3rd s., xxxvi, 97.

has been the favourite instrument, though recently paracentesis has several times been performed without any suction, by means of an ordinary trocar and cannula, and this is a simple and it appears a perfectly satisfactory method of operation in many cases.

Trephining of the sternum was a method suggested rather by theory than by practical experience. This operation introduces dangers of its own by exposing so vascular a bone as the sternum, and has now been entirely abandoned.

The place of punctures.—In thirty instances the fifth intercostal space was selected; in twenty the fourth; in three the sixth; in three the third; in one the seventh; in one the eighth. This last was a dry tapping. In most of the cases a spot was chosen as near as possible to the left edge of the sternum.

In three cases the puncture was made on the right side of the sternum; in one in the third right space; in two in the fifth right space.

The most convenient spot to puncture the pericardium was determined experimentally by Dieulafoy by the injection of fluid into the pericardium post-mortem. His observations showed that as the pericardium became distended, it stretched away in all directions from the base of the heart. The heart itself was gradually raised so as to be higher up than normal. In this way a considerable distance often intervened between the wall of the sac and the heart, even as much as two or three inches.

Dieulafoy recommended, therefore, the lowest convenient intercostal space, and, in order to avoid wounding the pleura, at no great distance from the sternum. These two conditions were best realized in the fifth space about one inch from the left edge of the sternum, and accordingly this is the place which he advocates for puncture. This is also the place which has been usually selected. The internal mammary artery is not at all likely to be wounded at this spot, for it has rarely been found more

than one third to one half of an inch from the edge of the sternum in this place.

Some recent experiments of Rotch,[1] by means of injection of cacao butter, has led this observer to recommend a very different place, viz. the right side of the sternum in the fifth space, about half an inch from the sternal edge. This he does upon certain theoretical considerations suggested by his cacao-butter injections. His conclusions I cannot think correct. They are directly opposed to Dieulafoy's and also to ordinary post-mortem experience. The position which the heart is found to occupy in the post-mortem examination of large pericardial effusions confirms the experiments of Dieulafoy, for in these cases the heart is found slightly displaced upwards, and the distended wall of the pericardium is at the greatest distance away from the heart walls at about the position of the normal apex. The danger of a wound to the heart is not very great, as has been shown many times by the results of injury and accident, and recently tapping of the right ventricle has been advocated in cases where it was greatly distended with blood, and has several times been performed with apparently good success. Whatever risk there might be could be avoided by carefully inserting the trocar, and sheathing it as soon as the heart was felt in contact with it.

When the pleura is obliterated and adherent to the pericardium the operation becomes much simpler. Then a spot may be safely selected as far out as possible, in the nipple line or even further, without any danger of opening the pleura.

From the preceding observations, the following conclusions may be drawn:

1. Paracentesis pericardii is not only justifiable, but an operation which may be safely undertaken with ordinary precautions. One case only is recorded in which the operation was in itself fatal. With this exception all

[1] 'Absence of Resonance in the Fifth Right Intercostal Space diagnostic of Pericardial Effusion,' by T. M. Rotch. Boston, 1878.

the patients were greatly relieved by the removal even of a small amount of fluid, and many recovered completely, who would probably have died if the operation had not been performed.

2. The most suitable place for puncture is, in ordinary cases, in the fifth left intercostal space one inch from the edge of the sternum, but if the pleura be adherent, the puncture may be made safely much further out, and even in the sixth space.

3. The instrument employed should be a trocar and cannula, with or without aspiration.

4. The operation may be performed with advantage not only in the pericardial effusions of rheumatic or primary origin, but also in those which occur in the later stages of general dropsy, if it should appear that the fluid in the pericardium is adding to the difficulties under which the heart is placed.

5. Purulent pericarditis is best treated on general principles, like an empyema.

a. The pericardial sac may in these cases be safely opened and drained.

b. This treatment moreover appears to be the only one which offers the slightest hope of recovery.

c. The results are likely to be more favorable than those of empyema, for the walls of the cavity are better able to contract rapidly and thus permit of complete obliteration.

Case.	Date.	Authos and reference.	Sex and age.	Clinical history.	Method and place of operation.	Nature and amount of fluid.	Result.	Post-mortem record.
19	1855	*Aran*, Trousseau, 'Clin. Med.,' iii, 391	M., 23	Patient had phthisis; parac. was performed twice, and a solution of iodine injected	Trocar in 5th left space: 1st puncture ..... 2nd puncture	 Serum—22 oz. Serum—49 oz.	R.	—
20	„	Do., do.	—	Both cases referred to by Trousseau, but no particulars given	—	—	R.	—
21	„	Do., do.	—		—	—	D.	—
22	„	*Vernay*, 'Gaz. hebd.,' 1856, 793	M., 23	Patient had valvular disease of the heart; was ill 1 month before parac. peric., and died 21 days after the operation, parac. abdominis being performed during this time	Trocar in 5th left space: 1st puncture ..... 2nd puncture	 Serum—500 cc. (17 oz.) Serum—400 cc. (14 oz.)	D.	Peric. contained "5 wine bottles full" of serum; peric. measured 25 cm. in diameter.
23	1856	*Trousseau*, 'Clin. Med.'	M., 27	Patient had phthisis, and had been very ill for 70 days before operation; after parac. effusion took place into left pleura, and 300 cc. were removed. Pat. died 5 days after operation, having had convulsive fits 3 days previously	Incision in the 4th left space, under the nipple; a gum sound was introduced through the incision	Serum—100 cc. (3½ oz.)	Patient relieved by operation D.	Peric. contained 1 litre of serous fluid; heart was 4″ away from the puncture; pleuritic effusion on left side.
24	„	*Bowditch*	—	Pepper (cf. Case 60) says it is doubtful whether this operation was completed	Incision	—	D.	—
25	1857	*Wileskowsky*, Gunther, utsupra	—	Scorbutus. Patient died 8 hours after operation	—	Blood	D.	—

		'Gaz. hebd.,' 1875, Nov.; 'Gaz. des Hôp.,' 1865	28	ill, completely recovered, and was in active work as a sailor 16 mos. later	space	greenish and cloudy		
27	1866	*Clifford Allbutt* and *Wheelhouse*, 'Brit. Med. J.,' 1868, Oct. 10	M., 26	Acute rheumatism. Patient left with a mitral murmur, but was well 23 mos. later	Trocar in 4th left space, ½ inch from edge of sternum	Serum—3 oz.	R.	—
28	1867	*Lovel & Mader*, 'Ctlblt.,' 1870, No. 3; 'Schmidt's Jahrb.,' 143, p. 137	F., 68	—	Needle and syringe, operation was performed twice, the needle on each occasion being inserted 3 times:		D.	Double hydrothorax; only a little pericardial fluid.
					1st puncture in 3rd left space	Serum—3 oz.		
					2nd puncture in 3rd right space	Serum—3 oz.		
29	„	*Baizeau*, quoted by Roger, 'Gaz. hebd.,' 1868, p. 515; 'Bull. de l'Acad. de Méd.,' Nov. 2, 1873	M.	Patient had been ill 17 days after an operation for the removal of a gland at the angle of the jaw. Air entered into the left pleura, the heart was wounded, and the patient died 2 hours after the operation	Skin incision, and trocar in the 6th left space, three fingers' breadth from the edge of the sternum	Venous blood—400 cc. (10½ oz.)	D.	450 cc. of blood in the left pleura, probably from the wound in the heart.
30	1868	*Roger*, 'Soc. Med. des Hôp.,' 1869	F., 12	Patient had phthisis, and lived 2 days after the operation	Trocar in 5th left space	Serum—780 cc. (27 oz.)	D.	300—400 cc. of bloody serum in peric.; tubercular pericarditis, myocarditis, and cardiac thrombi.
31	1869	*Clifford Allbutt* and *Teale*, 'Lancet,' 1869, p. 807	F., 27	Patient had phthisis, and died 8 hours after the operation	Trocar in 4th left space:		D.	—
					1st puncture	Serum—5 oz.		
					2nd puncture	Serum—6 oz.		

Case.	Date.	Author and reference.	Sex and age.	Clinical history.	Method and place of operation.	Nature and amount of fluid.	Result.	Post-mortem record.
32	1870	*Ponroy & Fremy*, 'Gaz. d. Hôp.,' 1870; 'Dieulafoy Aspiration,' 1873	M., 21	Patient had phthisis; had pericarditis and left pleuritic effusion; had been ill 14 days with pain in the left side	Aspirator 1 cm. above lower line of dulness, 6 cm. from middle line of sternum; canula pushed in 8 cm.	Sero-purulent—800 cc. (28 oz.)	R.	—
33	1871	*Malle*, 'Thèse de Labrousse,' 1871	—	Patient lived 8 days after parac.	Trepanning sternum, and injection of iodine	—	D.	—
34	1872	*Duncan*	M., 12	Patient lived 15 hours after parac.	—	—	D.	—
35	„	*Chaillon*, 'Dieulafoy Aspiration,' 1873	—	Patient had phthisis and pleuritic effusion; died 4 days after parac.	Aspirator	—	D.	—
36	„	*Chairon*, 'Bull. Acad. de Méd.,' 1872; 'Lancet,' 1872, Nov. 30	M., 23	Patient had phthisis and pleuritic effusion, 1430 cc. (52 oz.) removed; parac. peric. 2 days later. Patient had been ill 4 mos., and died 7 weeks after parac. peric. of diarrhœa	Aspirator in 5th left space, 2 cm. (1 inch) from edge of sternum	Serum—35 oz.	Relief. D.	Peric. contained 1000 cc. of serous fluid, tubercular; lungs tubercular.
37	„	*Maclaren*, 'Ed. Med. J.,' 1872, June	M., 27	Ill 4 weeks with pleurisy; died 6 days after operation	Skin incision, and trocar in 5th left space, close to edge of sternum	Serum—35 oz.	Relief. D.	Peric. contained 12 oz. of serum; pleura 2½ quarts of serum; lungs tubercular.
38	„	*Roger*, 'Bull. Acad. de Méd.,' 1875	M., 5	Patient admitted for rheumatic fever; had been ill for 4 mos. before with mitral disease. The heart was wounded during operation, but no ill effects followed.	Aspirator in 6th left space, 1 cm. (½ inch) under apex beat	Serum, then blood—200 cc. (7 oz.)	Relief. D.	Morbus cordis (mitral); adherent peric.

					Patient died of heart disease 6 mos. later				
	39	„	*Labric*, quoted by Roger	— 6	5 weeks ill with pain in the left side, thought to be pleural	Trocar in 5th left space, 2 inches inside nipple line	Pus—1000 cc. (35 oz.)	D.	Pleura adherent; 31 oz. of reddish serum in peric.
	40	„	*S. Gemell* and *Gairdner*, 'Glasg. Med. J.,' Nov., 1872	M., 27	Puncture caused great pain. Patient died 2 days after parac.	Aspirator in 6th left space, ½ inch inside nipple line; apparatus did not work well	—	D.	630 cc. (22 oz.) in peric.; pleuritic effusion; tubercles in pleura and peric.
	41	„	*Jürgensen*, 'Ziemmsen Handb.,' v, 134	M., 6	Patient had acute pneumonia for 14 days. Died 4 days after operation with brain symptoms	Aspirator: 1st puncture ..... 2nd puncture, 3 days later; 1% salt sol. injected	Pus—220 cc. (8 oz.) Pus	D.	Purulent peric. and meningitis.
	42	1873	*Heath*, *Barlow*, *Ringer*, 'Practitioner,' xi, 265	M., 6	Patient had phthisis, and later, ascites, which was tapped. Died 7 mos. after commencement of illness, and 2½ mos. after first parac. peric.	Aspirator: 1st puncture in 4th left space, 1 inch below, and ¾ inch inside, left nipple 2nd puncture in 5th space 8 days later	Serum—3½ oz. Reddish-brown serum—6 oz.	D.	Tubercular peritonitis; peric. contained half a pint of straw-coloured fluid.
	43	„	*Bouchut*, *Danlos*, 'Gaz. d. Hôp.,' 1873, 142	F., 11	Patient had pleuritic effusion on left side for 6 weeks; perac. peric. 8 times. Death 3 days after the last, 34 days after the first parac. Heart wounded twice without bad result	Trocar in 5th left space, 1½ in. from edge of sternum: 1st puncture ..... 2nd puncture ..... 3rd puncture ..... 4th puncture ..... 5th puncture (heart pricked)..... 6th puncture ..... 7th puncture ..... 8th puncture (air entered).....	Serum .....320cc.(11½oz.) Reddish do. 510cc.(17½oz.) Bloody do. 150cc. (5 oz.) Dry ..... — — Bloody do. 220cc. (8 oz.) Bloody do. 150cc. (5 oz.) Chocol. do. 330cc.(11½oz.) Dry ..... — — Total...1680cc.(58½oz.)	D.	800 cc. of chocolate coloured fluid in peric.

Case.	Date.	Author and reference.	Sex and age.	Clinical history.	Method and place of operation.	Nature and amount of fluid.	Result.	Post-mortem record.
44	1873	*Villeneuve*, 'Gaz. Med.,' 1875	M., 5½	Traumatic peric. after a fall; œdema of skin and fluctuation; after the parac. a fistula formed in the track of the puncture, which discharged serum, and later pus, for 5 months. At the end of 6 mos. the fistula closed. Patient was well 10 mos. later	Trocar in the prominent part of the swelling	Serum—2 syringefulls	R.	—
45	1874	*Saundby*, 'Ed. Med. J.,' 1875	M., 13	Patient had been ill 7 days with pleurisy; died a few hours after parac. peric.	Aspirator in 4th left space, ½ inch from edge of sternum	Pus—30 oz.	D.	Purulent pericarditis; peric. contained 36 oz. of pus; it was villous and adherent posteriorly; small abscesses in lower lobe of right lung.
46	„	*Bartlett*, 'Lancet,' 1874, 866	M., 20	Patient had been ill with rheumatic fever 14 days; had old mitral disease and recent left pleuritic effusion. Complete recovery in 4 weeks, the fluid in both peric. and pleura being absorbed	Aspirator in 4th left space, 2 inches from middle line of sternum	Bloody serum—14 oz.	R.	—
47	„	*Lyon*, 'New York Med. Rec.,' April, 1867	M., 31	Patient died 16 days after the parac. peric.	2 punctures	Pus	D.	—

48	„	*Ferrari-Bravo* and *Valtosti*, 'L. Med. Rec.,' iii, 1875	M., 35	Parac. of the peric. pleura and abdomen. Patient had phthisis	Skin incision, and aspirator in 5th left space, $\frac{2}{5}$ inch from sternum	Serum—10 oz.	D.	Peric. much thickened, containing 3 pints of serum; right lung tubercular; left compressed by pleuritic effusion.
49	„	*Shingleton-Smith* and *Steele*, 'Lancet,' 1874, 271	M., 25	Patient had had rheumatic fever four times; had been ill 9 mos. Died 67 days after parac.	Trocar in 4th left space, midway between mid-sternal line and the left nipple	Bloody serum—10 oz.	R.	—
50	„	*Macleod Gairdner*, 'Glas. Med. J.,' 1877	M., 23	Patient had been ill 30 days after getting wet. Peric. was tapped 3 times. Patient died 3 days after the last, 28 days after the first parac.	Trocar in 5th left space, 2 in. from edge of sternum: 1st puncture ..... 2nd puncture (10 days later)..... 3rd puncture (11 days after 2nd)...	Bloody serum—20 oz. Do. 30 oz. More blood — 15 oz.	D.	—
51	„	*Andrew Callender* (not published); record in St. Barth. Hosp. Notes	M., 14	In course of rheumatic fever	Trocar and cannula in 4th left space, near edge of sternum	Blood—2 oz.	D.	5 min. after puncture from hæmorrhage into peric.; right ventricle torn by trocar about middle of ant. surface, close to ordinary position of the white spot.
52		*Sir Risdon Bennett* (not published)	M., 20	Supposed to be a mediastinal cyst. Case under observation 4 years and 3 mos. Was tapped many times with great relief, and temporary recovery. P.M. showed chr. peric. effusion	Many punctures	Clear serous, once containing cholesterin. Large quantity on each tapping	—	Chr. peric. effusion; acute tubercular pericarditis and peritonitis, with chr. adh. peritonitis.

Case.	Date.	Author and reference.	Sex and age.	Clinical history.	Method and place of operation.	Nature and amount of fluid.	Result.	Post-mortem record.
53	1875	*Moore, Gooch, G. Johnson,* 'Brit. Med. J.,' 1874	M., 13	Patient died 38 days after parac. from diarrhœa	Trocar in 5th left space, just inside the normal position of the apex beat.	Pus	D.	1 pint of pus in peric.; fatty heart; abscess in thigh; peritonitis.
					1st puncture	21 oz.		
					2nd (5 days later)	35 oz.		
					3rd (7 „)	60 oz.		
					4th (10 „)	50 oz.		
					5th (3 „)	30 oz.		
					6th (3 „)	9 oz.		
					Iodine sol. injected after the last 3	Total 205 oz.		
54	„	*Elliott, Burder,* 'Lancet,' 1876; 'Trans. Bristol Med.-Chir. Soc.,' 1878, i, 75	M., 60	Patient had rheumatic fever; was made O.P. 9 weeks after parac.	Trocar in 5th left space, 1 inch inside left nipple line	Serum—42 oz.	R.	—
55	„	*Exchequet, Polain,* 'Bull. Soc. Anat.,' 1875	M., 60	? Traumatic pericarditis after a fall	Trocar in 8th left space	Dry	D.	2 pints of seropurulent fluid in peric.
56	1876	*Nixon,* 'Dubl. J. of Med. Sc.,' 1876	M., 20	Patient had rheumatic fever, with pleurisy and congestion of lungs. Died 6 days after parac.	Skin incision and aspirator (No. 2 Dieulafoy, and then No. 4) in 5th left space, 1¼ inch from edge of sternum	Bloody serum—3 oz.	D.	30 oz. of seropurulent fluid in peric.
57	„	*Welch,* 'Amer. J. of Med.Sc.,' 1877	—	Patient lived a few days after parac.	Aspirator	Pus—28 oz.	D.	—

58	1877	*Hunt, Wilson Fox*, 'Lancet,' March, 1877	F., 16	Rheumatic fever (2nd attack)	Aspirator in 5th left space, close to sternum	Serum and a little blood—½ oz.	D.	12 to 14 oz. of serous fluid in peric; vegetation in mitral valve.
59	1878	*Steel*, quoted by Wheelhouse, 'Med. T. and G.,' 1878, 191	M., 35	Chronic bronchitis. Patient much relieved by parac.	Aspirator	Serum—4 oz.	R.	—
60	1879	*Pepper, Rex*, 'Amer. J. of Med. Sc.,' 1879, April	F., 17	Patient had albumen and casts in urine, which disappeared after operation. Patient much relieved by parac.; could walk about at end of the month. Died suddenly with ascites 3½ months later	Aspirator in 5th left space, 1 inch inside nipple line	Reddish serum—8 oz.	Relief. D.	Adh. peric; granular kidney; ascites.
61	„	*Reisz & Levison*, 'Hosp. Tidend,' 1879, 2 R., vi	M., 10	Patient had been ill for 4 weeks with recurrent pneumonia; had 3 attacks before parac., and one after, which proved fatal	Aspirator in 5th left space	Pus, blood-stained—"considerable quantity"	D.	—
62	„	*Hindenlang*, 'Deutsch Arch. f. Klin. Med.,' 1879, 452	M., 20	Case of acute pericarditis of 2½ mos. duration before treatment. Well marked pulsus paradoxus, with cyanosis. Parac. performed 3 times. Marked increased in urine after operation. Patient was discharged in 6 mos. still cyanotic. Died 9 mos. after operation	Aspirator. 1st, several punctures in 5th left space, 5 cm. (2″) from sternum ... 2nd, in 5th space, 6 cm. from sternum (2½″), next day... 3rd, in 4th space, 2 cm. from sternum (1″).....	 Failed Serum—300 cc. (10½ oz.) Serum—500 cc. (17½ oz.)	R.	—

Case.	Date.	Author and reference.	Sex and age.	Clinical history.	Method and place of operation.	Nature and amount of fluid.	Result.	Post-mortem record.
63	1879	*Hindenlang* and *Kussmaul*	M., 21	Patient had right pleuritic effusion; puncture was made from right side, but as it drew off two kinds of fluid on pushing needle deeper, K. thought the peric. had been tapped as well as the pleura. Patient quite well 7 mos. after operation	Aspirator in 5th right space, 1½ cm. from nipple line:		R.	—
					1st puncture	Serum—750 cc. 26 oz.); uncertain how much came from pleura		
					2nd puncture	550 cc. (19 oz.) from pleura 250 cc. (9 oz.) from peric.		
64	„	*McCall Anderson*, 'Glas. Med. J.,' 1879, 214	M., 17	—	Aspirator in 5th left space, 1 inch from nipple line	Serum—38 oz.	R.	—
65	„	*Viry*, 'Union Médic,' 1879, 3 s., xxvii, 315	M., 22	Pneumonia, right base. 1 week later pericard.; no friction. 7 days later parac. Next day parac.; cannula left in. Died 12 hours later	Aspirator, trocar, and cannula.	Pus—	D.	Traces of pneumonia; purulent peric.; peric. villous, pouched, containing 2 to 3 litres of pus.
					In 5th left space, 3 cm. from edge of sternum	43 cc. (1½ oz.)		
					Two punctures ...	140 cc. (5 oz.)		
66	1880	*P. Kummell*, 'Berl. Klin. Wochsch.,' 1880, No. 23	M., 22	Rheumatic fever (2nd attack)	Aspirator in 4th left space	Bloody serum—2 oz.	R.	—
67	„	Ditto	M., 50	Effusion into left pleura	Aspirator in 4th left space, 4 cm. from edge of sternum:	Serum—	D.	Peric. partially adherent, containing 35 oz. of serum.
					1st puncture	27 oz.		
					2nd puncture	30 oz.		
68	„	*Richard*, 'Rec. d. Mem. d. Med. Milit.,' 1880, 3 S., xxxvi, 97	M., 28	Patient had been ill 1 month before operation; had left pleurisy after, and died 6 weeks after parac.	Aspirator in 4th left space, 5½ cm. (2 inches) from edge of sternum	Reddish-brown serum—1180 cc. (41 oz.)	Relief. D.	Tubercular pericarditis; peric. contains 2100 cc. (75 oz.) brown, thick, almost oily fluid

69	„	*Gee, Steavenson*, 'St. Barth. Hosp. Rep.,' 1881	F., 5½	Patient was ill 3½ mos. with left empyema, which was tapped twice (21 oz. and 3 oz.). Died 14 days after parac. peric. which gave great relief	Trocar in 3rd left space, ⅔ inch from sternum	Serum—5½ oz.	Relief. D.	Empyema; chronic pericarditis.
70	„	*E. Wagner*, 'Berl. Klin. Wochens.,' 1881, 361	M., 12	Strumous child. Great relief from operation. Died 14 days later	Trocar. 1st puncture to right of sternum, because the dulness extended so far ..... 2nd to left of sternum..... 3rd, 14 days later	Serum—Little „ 6 oz. „ —	D.	Tubercular pericarditis; tubercles in lungs and pleura.
71	1881	*Fiedler*, 'Jahrb. d. Ges. f. Nat. Heilk. in Dresden,' 1881, 137	F., 25	Patient had general renal dropsy, which disappeared after parac.; great increase in urine after operation. Patient left well, and was afterwards a nurse in a lunatic asylum	Trocar	Serum—1100—1200 cc. (40 oz.)	R.	—
72	„	Ditto	M., 25	Rheumatic fever. Preliminary exploration with injecting syringe; increase of urine after operation	Trocar. 1st puncture ..... 2nd (8 days later)	Pus— 800—1000 cc. (30—35 oz.) 700 cc. (25 oz.)	D.	Purulent pericarditis; cor. villosum; recent vegetations on aortic and mitral valves.
73	„	Ditto	M., 30	5 weeks ill with right pleuritic effusion. Preliminary exploratory puncture. Increase of urine after operation	Trocar. 1st puncture ..... 2nd (3 days later)	Pus— 800 cc. (28 oz.) 1500 cc. (52 oz.)	D.	Purulent pericarditis; caries of 8th & 9th dorsal vertebræ, with abscess under pleura. No connection with peric. found.

Case.	Date.	Author and reference.	Sex and age.	Clinical history.	Method and place of operation.	Nature and amount of fluid.	Result.	Post-mortem record.
74	1881	*Hunt*, 'Lancet,' 1881, May	M., 23	Patient had aortic disease; peric. friction developed. The trocar wounded the heart and was withdrawn; no harm was done	Trocar in 4th left space, near the sternum	0	D.	Peric. adherent in places, but contained a large amount of fluid.
75	„	*Pitts, Harley* (not recorded), from St. Thos. Hosp. Notes	F., 28	Rheumatic fever 1 mo. before admission; dyspnœa 1 week. Melancholia, with delusions, during convalescence. Left 4 mos. later quite well, but had been employed for some weeks previously as a scrubber in the hospital	Skin incision and aspirator in 5th left space, 1½ inch from edge of sternum	Bloody serum, which coagulated immediately—9 oz.	R.	—
76	„	*Rosenstein Iterson*, 'Berl. Klin. Wochsc.,' 1881, xviii	M., 10	After twice tapping the peric. a free incision was made, which gave exit to a very large quantity of pus; operation was antiseptic, and drainage tube inserted. Discharge lasted 68 days, during which time the left pleura was laid open for empyema. 1100 cc. and 1000 cc. had been removed by parac. previously	Trocar in 4th left space, close to edge of sternum. 1st puncture ..... 2nd puncture ..... Free incision	Pus— 620 cc. (22 oz.) 120 cc. (4 oz.) Large quantity	R.	—
77	„	*Douglas Powell*, from private notes	M., 17	3 mos. ill from "inflammation of the lungs." Died 3 days after ope-	Aspirator, after preliminary exploration, in 5th right	Bloody serum—17 oz.	D.	Dense-walled pericardium; sac contained 25 oz.

		at Brompton Hospital		ration	space, ½ inch from edge of sternum			of blood-stained serum.
78	1882	*West*	M., 16	Pain in left side for 3 weeks; pulsus paradoxus; cyanosis. Parac. performed twice; then free incision; drainage tube inserted. Discharge free for 10 days; wound closed on 30th day; patient up on 36th day. Complete recovery	Trocar. 1st puncture in 4th left space, under nipple ..... 2nd puncture in 5th left space ... Free incision	Pus. 16 oz. 13 oz. Large quantity	R.	—
79	,,	*Clifford Allbutt, Ward, Silk,* 'Lancet,' Jan. 27, 1883	M., 20	? Rheumatic fever. Ill 6 weeks. Had effusion 1 month before parac. Great relief by operation; rapid absorption of fluid afterwards. Complete recovery	Aspirator in 4th left space, 2½ in. from middle line of sternum	Clear serous fluid—1 oz.	R.	—
Recent case not included in present paper.								
80	1883	*Savory.* Recorded in St. Barth. Hosp. Notes, but not yet published	—	Pyæmia after a severe fall; abscess in shoulder. Left pleurisy, tapped twice. Free incision made into pleura in 5th left space in anterior axilla; no fluid found, but peric. felt to be distended and opened from incision in pleura. Temporary relief. Died 14 days later. Pulsus paradoxus	Incision made from pleural incision	Pus—24 oz.	D.	Scar of abscess over left shoulder; large abscess in thigh; three loculated left pleural effusions, one only tapped. Peric. empty, but adherent, except at lowest and outermost part, where surface of heart was covered with flaky lymph.

Case.	Date.	Author and reference.	Sex and age.	Clinical history.	Method and place of operation.	Nature and amount of fluid.	Result.	Post-mortem record.
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76	„	*Rosenstein Iterson*, 'Berl. Klin. Wochsc.,' 1881, xviii	M., 10	After twice tapping the peric. a free incision was made, which gave exit to a very large quantity of pus; operation was antiseptic, and drainage tube inserted. Discharge lasted 68 days, during which time the left pleura was laid open for empyema. 1100 cc. and 1000 cc. had been removed by parac. previously	Trocar in 4th left space, close to edge of sternum. 1st puncture ..... 2nd puncture ..... Free incision	Pus— 620 cc. (22 oz.) 120 cc. (4 oz.) Large quantity	R.	—
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GLIOMATOUS ENLARGEMENT OF THE PONS VAROLII IN CHILDREN.

BY

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(Received April 10th—Read May 8th, 1883.)

I WISH to place on record two more cases of "gelatiniform enlargement of the pons Varolii." Readers of the thirteenth and seventeenth volumes of the 'St. Bartholomew's Hospital Reports' will find a case of this disease described in each volume—one by Dr. Percy Kidd and the other by Dr. Gee, though both were under the care of the latter physician during life. I had the good fortune to be charged with the microscopical examination of the specimen, of which the clinical account may be found in the seventeenth volume of the 'Reports.' Concerning the affection Dr. Gee wrote thus: "So far as I know, the disease has been hitherto undescribed." This statement is the chief cause of the appearance of the present paper.

My special thanks are due to Dr. Gee for primarily directing my attention to the disease, which I need hardly say was entirely new to me.

Case 1.—Alfred James B—, æt. 11, was admitted into the Hospital for Sick Children in November, 1873, under the care of Dr. Dickinson. His illness began three months before admission, when it was noticed that the head was held to the left side, that there was loss of control over the left foot, that the left hand dropped; he had vomited especially in the morning, had grown dull, and walked badly. The bowels were said to have been regular. There was some complaint of headache at the postero-dextral region. There had been otorrhœa on the right side since the child was six months old. The patient had had a cut on the head four years ago. The father's grandmother died insane. No alcoholism. He was one of ten children, of whom two had epileptic fits, one died of "brain fever," and one died of "fever," and had otorrhœa. There seemed to be no evidence of struma, syphilis, or tubercle. The patient was said to have always been excitable.

State on admission.—Well nourished. Holds head to left shoulder. Deaf of right ear from which a discharge of matter issues. Can stand and walk, but drags left leg. Grasp of left hand weaker than right. Sensation diminished in left arm and leg. The left calf measures 9 inches, the right 9⅛ inches. The bowels and bladder act naturally. Bowels are confined. Vomiting. Left pupil dilated and sight is worse in the left eye. Ocular movements perfect and equal. Sensation and motion of face perfect. Tongue protruded in mid line.

On November 14th the patient was more drowsy and the intellect was duller.

21st.—Complains of feet feeling cold, especially of the left.

27th.—Pain in back of head and giddiness were noted.

28th.—Giddy but no headache.

December 7th.—Left optic disc too pink, outline distinct, vessels not very tortuous nor dilated; right optic disc natural.

26th.—Further loss of power in left arm and leg, cannot draw left leg up to abdomen without the help of his hand. Grasp of left hand much weaker than right. Other symptoms continue.

January 15th.—Has lost all power in left hand; can only move left arm a little; thumb is turned into palm, and fingers are flexed thereon. Only slight voluntary power over left leg. Food lodges between gums and left cheek; there is constant dribbling.

February 4th.—Face often flushes. Cannot swallow so well. Speaks indistinctly. Abdomen retracted.

6th.—*So weak and faint* that death was hourly expected. Mucus on both eyeballs; takes but little notice when shouted at. Left arm and leg are rigid. Left ear discharging. Sordes on teeth. Dirties and wets bed.

10th.—Bedsore on left ischial tuberosity. Unconsciousness. Less otorrhœa.

12th.—More conscious. More otorrhœa.

24th.—Conscious. Bowels not open for eighteen days.

March 11.—Boy continues to be sensible. No action of bowels for thirty-five days.

24th.—Left arm rigid and helpless. Right arm less rigid (no previous note of rigidity here), some power in it; can move right but not left leg.

29th.—Death.

Post-mortem (thirty-five hours).—Weight of brain 45¾ ounces. No sign of inflammation anywhere in cranium. There was enormous expansion of the pons Varolii and crura cerebri. The "bridge" measured 2 inches from side to side, 1⅞ inches from before back, or about half as much again as natural for that age. Dr. Dickinson said that in shape it was what mineralogists call "botryoidal," and resembled in colour, &c., "chalcedony;" further, it looked like a "soft package tightly corded." The depressions corresponding to blood-vessels. The basilar artery lay in a deep furrow. There was a great prominence at the anterior part of the left side of the pons, behind the root of the third nerve; being greatest between the third

and the fifth nerve. The cerebral peduncles were swollen, the right the more; also both cerebellar peduncles. Both third nerves were much flattened. The medulla oblongata was natural. The tuba cinereum expanded. The arachnoid on the convexity of both hemispheres was thickened and opaque. There was about four ounces of fluid in the lateral ventricles. Some pneumonia of the right lung. The spinal cord was natural.

For the use of the above notes my thanks are due to Dr. Dickinson. As the microscopical examination is precisely like that found in the other cases, a single description will serve for all, and this will be given later. A drawing made by Dr. Westmacott gives a fair representation of the pons and neighbourhood of this case (see Plate IX).

Case 2.—Emily H—, æt. 6½, admitted into the Hospital for Sick Children on December 18th, 1882, under the care of Dr. Cheadle, who has kindly given me permission to make use of the case. Her illness began about four weeks before entry, with occasional vomiting, with headache, and much giddiness, and some degree of languor; the gait had been noticed to be staggering for the space of three weeks, and a squint had been observed for two weeks. She is said to have been "convulsed." She was a studious child. No cause could be given for the illness. The patient had had measles at the age of three years, this was followed by hooping cough, and at the age of four years and a half scarlet fever was said to have affected the patient. She had never had teething convulsions. The family history showed that there had been eight children with two miscarriages of no special relation to one another, and not in any way suggestive of syphilis. There were seven children living, the seventh out of eight had died from "effusion into brain" at the age of eleven months. Emily H— was the sixth child. Some of the children had thrush for two weeks from birth, but no snuffles. There was no history of rickets or consumption; some of the children had had an abscess in the neck.

The father's brother was supposed to have died of consumption; he was a soldier. The father had never been in the army or navy and denied all forms of venereal disease. The mother has been and is still suffering from "rheumatic iritis" for four years.

On December 21st the present state was taken, the patient having been watched since entry. The girl was rather drowsy, did not play, and remained quietly lying down all day. She had vomited once and thrice on the 19th and 20th respectively. The temperature was 99·2° in the evenings (about 6 p.m.) and about 98·4° in the mornings (about 10 a.m.). Pulse 132, regular (several observations). Breathing 24, regular. Urine acid, no albuminuria, no glycosuria, no increased frequency of micturition, the amount, however, was not measured. Bowels constipated, not open for two days. The belly was not full, the lower edge of the spleen was palpable.

Sensation and special senses were apparently natural, but this examination was difficult to carry out owing to the patient's impaired mental state. The speech was decidedly altered, being of a spasmodic character. Walking was just possible with support, but the gait was very jogging and unsteady; the patient could not stand alone. There was a convergent squint; the eyeballs moved about in every direction except outwards. There was probably paralysis of both external recti. The pupils were of medium size, equal, and acted, but whether to accommodation as well as to light could not be ascertained. Mr. Marcus Gunn detected some abnormality in the shape of a spot situate to the inside and rather above the right optic disc; there was a little hypermetropia, no optic neuritis. The muscles of mastication seemed to act naturally. There was no paresis of either half of the face. The tongue was protruded fairly well and symmetrically, had many large papillæ, and was rather dry. The patient had a stupid look, the mouth was half open, there was no dribbling of saliva. When the patient sat up in bed, which act she was just capable of performing, a coarse

indefinite tremor was observed to affect the trunk and head. Micturition and defæcation were performed without calling for the nurse. The knee phenomena were present and equal, and easily obtained. Nothing definite could be learnt from the superficial reflexes, the patient became so restless under examination. Nothing abnormal made out in the heart or lungs.

December 23rd.—Pulse 108 and regular.

26th.—The temperature was 100·6° on the night of the 21st, otherwise it had not been above the normal. The notes then say that patient got quite excited at times, and that she had narrated her experiences of the performances of the Salvation Army; that, although drowsy and quiet for the most part, she was communicative at times, the speech being jerky and peculiar. There had been no fresh vomiting.

29th.—All the symptoms have continued; the edges of the optic discs are obscure, nothing wrong noticed in the vessels of the fundus oculi. The knee phenomena are certainly excessive this morning, but no ankle clonus could be got. There has been vomiting the last three days.

30th.—The patient got very drowsy about 4 p.m. The respiration began to be very slow, whilst the heart was going fairly well. The pulse was 84, and regular. Much vomiting. She had to be fed through the nose. The coma endured and the breathing failed at 1 a.m., December 31st.

Autopsy (twelve hours after death).—Rigor mortis everywhere present. There were no signs of rickets, syphilis, or struma. The meninges and large vessels were natural. The pons Varolii was enlarged in all its dimensions, the left side perhaps more than the right, the cerebellar peduncles were enlarged, especially the left one; the basilar artery lies in the valley formed by the overgrowth of each half of the pons.

The greatest width was 2¼ inches.
,, length ,, 1½ ,,
,, thickness ,, 1¼ ,,

The left cerebral peduncle was perhaps a little enlarged. Both the third nerves were pressed upon. The fifth left nerve was enlarged at its superficial root. The medulla oblongata seemed swollen, but its parts were recognisable. The floor of the third ventricle was convex and too prominent. The sixth and seventh nerves were involved at their superficial origins. The other nerves were apparently free from disease at their superficial roots. Sections show the pons to be diseased right through its substance, and in the centre there was a grey-red area. The whole of the floor of the fourth ventricle seemed to be affected with the disease. There were three flame-shaped hæmorrhages in the right retina, disposed in a radiating manner in the neighbourhood of the optic disc; they were not more than three lines long. Both optic discs had a swollen look, the vessels were not abnormal. There were no hæmorrhages into the left retina. Both internal ears were natural. There were genuine ecchymoses, not mere hyperæmia, in the gastric and intestinal mucous membranes, a few patches in the small bowel and one in the cæcum. The kidneys were sanguinolent, and their consistence was increased, they weighed 4½ oz. together. The spleen was flabby, weighed 3¼ oz., had very abundant white areas, "Malpighian corpuscles," so that the intervening red tissue formed only a fine network as seen on section. The liver weighed 21 oz., was full of blood, and had its consistence somewhat increased. None of the above-mentioned organs gave any reaction with iodine. No tubercle was found anywhere. The other organs, including the lymphatic glands, were all natural. Some urine, tested from the bladder, was acid, had no albumin, and no sugar.

Microscopical characters.—The most obvious change is a small-celled infiltration; sections from the thick part of the tumour consist of very little else than cells. The shape of these units is various, they are mostly round or oval, sometimes reniform. The alteration of the brain at this part is so great that it is impossible to recognise any

normal structure. A stained section examined with the naked eye by transmitted light shows certain markings, which in their general arrangement recalls the naked-eye appearance of the pons. Such a recognition of normal configuration is very striking. This replacement of healthy structure by diseased tissues without much alteration of the macroscopic appearance is a fact of not unfrequent occurrence, not only in the brain but in other organs. The normal anatomy draws the lines along which future disease must be built. This may be called the "principle of the preservation of pattern." The neuroglia and natural nerve-fibres are not recognisable. There is an intercellular substance of homogeneous appearance which does not become black when treated with osmic acid. I conclude from this that normal medullated fibres are not present; though the presence of axis cylinders is of course not denied. No nerve-cells are to be detected where the growth is most abundant, but in places a ganglion cell very little changed from the healthy appearance may be discovered. There is nothing to be said of the state of the blood-vessels. There are no extravasations.

My thanks are due to Dr. Gowers for having examined my sections. He fully agreed with my interpretation of them.

Remarks.—It seems certain that till now little or nothing has been known of such a disease as nearly symmetrical gliomatous enlargement of the pons Varolii by the majority of physicians. The evidence for that opinion consists in the facts that Dr. Dickinson described his example as one of "peculiar hypertrophy," &c., and that Dr. Gee recorded his cases and wrote of them in the sentence which has already been quoted. Further, compared with my own sections, Dr. Percy Kidd's specimens, which he has kindly given me the opportunity of examining, are undoubted samples of glioma, and thus there should be no necessity for the continuance of the name 'gelatiniform enlargement of the pons." That phrase has served its purpose

and may now be eliminated from medical nomenclature. The macroscopical aspect of the affection is by far the most striking and interesting feature, and for this reason a plate taken from a drawing by Dr. Westmacott has been added to this paper (see Plate IX). With regard even to the coarse etiology, we are very much in the dark. It would appear that traumatism has been observed to precede the appearance of the disease in the pons as of other parts of the brain, but the influence of injury in the causation must be a very difficult point to settle. The disease seems to have its starting-point in the neuroglia, and hence we can partly understand the insidious mode of its clinical commencement and progress. The signs during life are frequently more marked on one side of the body; this was so in the first case here recorded. Gee, Kümmell, and Leyden have reported similar cases ('St. Barth. Hosp. Rep,' xvii, and 'Zeitschrift für Klin. Mediz.,' Band. ii, S. 282, 1881).

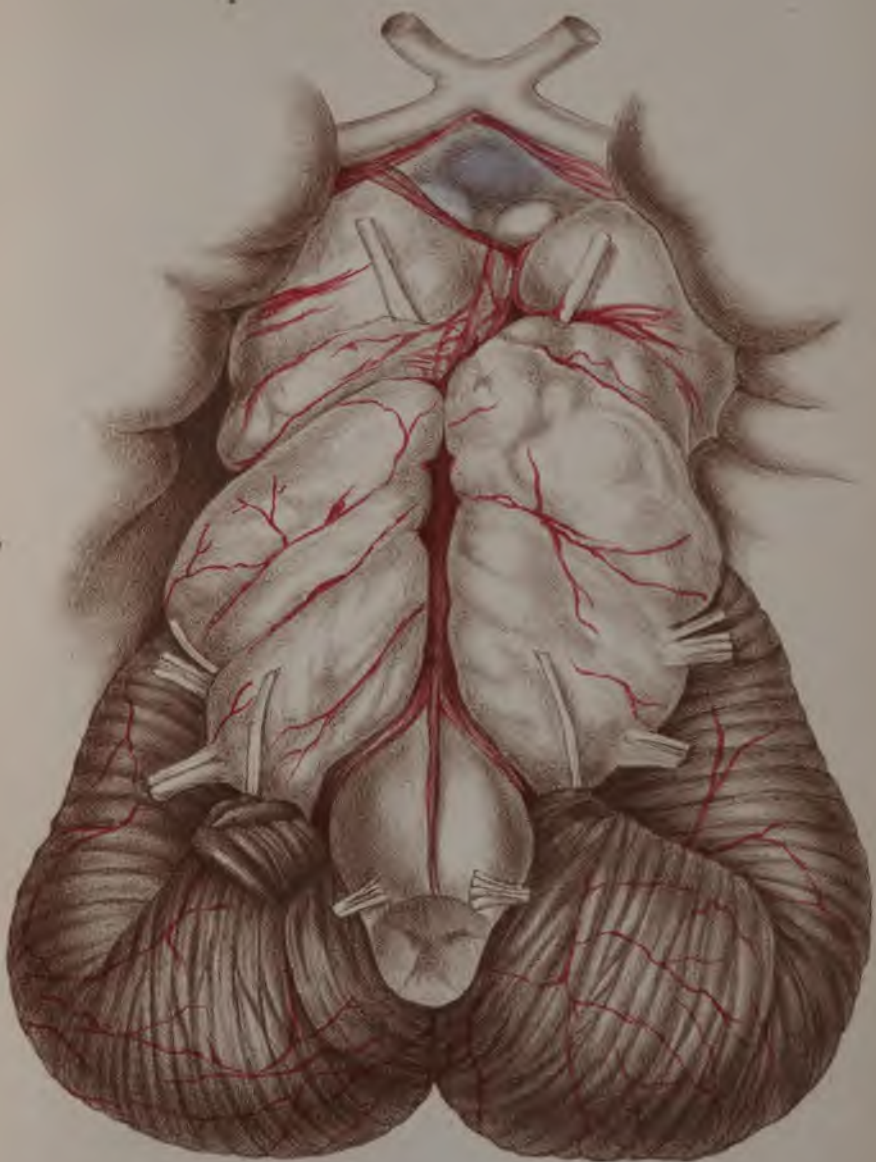
I have nothing to add to what is already known about the pathological and regional diagnosis. Polyuria has been observed but never albuminuria or glycosuria, nor has any sudden elevation of the temperature of the body been recorded, such changes are more likely to occur in acute affections of the pons (see Gowers in second volume of 'Brain,' p. 466). Though it is conceivable that a hæmorrhage might cause sudden pyrexia or glycosuria. And hæmorrhages are liable to occur in such gliomata.

After writing the above, my attention was directed by Dr. Gowers to a paper by Schulz in the 'Neurologisches Centralblatt' for January, 1883. This article narrates a case of the disease in a man, æt. 32, which is precisely similar to my cases in the symmetrical enlargement of the pons and other characters. Schulz has also collected all the cases recorded of glioma of the pons, whether localised or diffuse; the majority occurred in children not over the age of twelve. Lastly, Schulz boldly records Kidd's

case ('St. Barth. Hosp. Rep.,' xiii) as a sample of glioma, though it is probable that he has never seen Dr. Kidd's specimens.

DESCRIPTION OF PLATE IX.

(Gliomatous Enlargement of the Pons Varolii, ANGEL MONEY, M.D.)



J.F. Westmacott ad Nat.m Del.t

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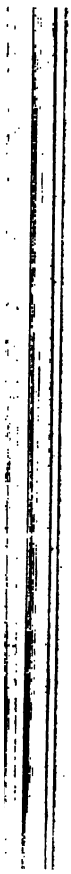


A CASE

OF

ASYMMETRY OF THE BRAIN

PRESENTING PECULIARITIES WHICH BEAR UPON THE QUESTION OF THE

CONNECTION BETWEEN THE OPTIC NERVES AND CERTAIN DEFINITE AREÆ OF THE CEREBRAL CORTEX.[1]

BY

SEYMOUR J. SHARKEY M.A., M.B., Oxon.,

ASSISTANT PHYSICIAN AND JOINT LECTURER ON PATHOLOGICAL ANATOMY AT ST. THOMAS'S HOSPITAL.

(Received April 10th.—Read May 8th, 1883.)

E. A—, æt. 25, was admitted into St. Thomas's Hospital under the care of Mr. Mason, owing to injuries received by the fall of a house. She died of the results of the accident in a manner which need not be related now, as it has no bearing upon the condition of the brain found after death, which is the subject of the present communication. The condition referred to was evidently a congenital abnormality, and was accompanied by a certain diminution in size of the right arm and leg, and by rigidity of the former.

I never saw the patient until I performed the post-mortem examination upon her, and no account was obtained

[1] The specimen is now in the Museum of St. Thomas's Hospital.

during her lifetime of the origin of the abnormality in the right arm and leg, nor of the state of her mind and special senses. The scraps of information which have since been received can pretend therefore to no scientific accuracy. But even so, the brain presents peculiarities which make it an anatomical specimen of very unusual interest, and which may contribute to the elucidation of certain points in the physiology of the cerebral hemispheres.

After her death the patient's father informed me that his daughter had always been healthy up to the time of the accident, which proved fatal in a few weeks; that she had never suffered from fits or other nervous affections; that from her very birth she had had a small and stiff right arm; and that she had always been left-handed and a little lame. He said that he had never noticed any mental deficiency, and that she had never complained of her sight, hearing, or other senses.

No peculiarity in her eyes, speech, or aspect, was noticed during her stay in the hospital, but the sister of the ward says that she struck her as being intellectually dull, even for a person in her position of life.

With these few remarks I shall pass on to the results of the post-mortem examination.

Both the right arm and leg were evidently smaller than their fellows though they were otherwise normally formed, and the muscles examined presented the rich brown colour of health. The right wrist measured $4\frac{3}{4}$ inches, the left $5\frac{3}{8}$ inches; the right forearm $6\frac{1}{2}$ inches, the left 8 inches. The circumference of the left leg round the calf exceeded that of the right by one inch. The heart and lungs and other viscera were healthy, with the exception of the kidneys and the bladder, which were in a state of acute inflammation. The skull and the membranes of the brain were normal, as were the intracranial blood-vessels, nor was there any marked inequality of the latter on the two sides.

On examining the brain it was seen at once that the

left hemisphere was smaller than the right, but that the difference in length was less than the difference in breadth and general bulk. One of the characters which specially struck the observer's eye was that laterally the most prominent part of the right hemisphere corresponded with the region of the supra-marginal and angular convolutions, whereas in the left the brain was quite flat in this position. The measurement of the hemispheres from the tip of the frontal lobe along the margin of the great longitudinal fissure to the tip of the posterior lobe was on the right 8½ inches, on the left 8 inches. The circumference, from the edge of the great longitudinal fissure over the parietal lobe to the internal border of the temporo-sphenoidal lobe on the under surface of the brain, was on the right 6⅞ inches, on the left 5⅞ inches.

On closer examination it became evident that most of the convolutions of the right hemisphere had their representations on the left, though the latter were smaller than the former. There was, however, one very striking exception. The angular and superior temporo-sphenoidal convolutions on the left appeared to be absent, and the supra-marginal convolution was represented by two thin strips of brain substance, which ran vertically upwards from the edge of the fissure of Sylvius, and lay between the posterior extremity of the latter and the base of the ascending parietal convolution. In the right hemisphere the angular and superior temporo-sphenoidal convolutions were well developed. The second annectent convolution on the right joined an offset from the angular gyrus and so formed a convolution which became continuous with the middle temporo-sphenoidal. But on the left the convolution which ran into the upper temporo-sphenoidal convolution was formed by an offset from the second annectent and another direct from the superior parietal lobule, the lower border of the latter owing to the absence of the angular gyrus coming to within one sixteenth of an inch of the upper extremity of the fissure of Sylvius, while on the right it was separated from this fissure by

about one inch and a quarter, a space occupied by the highly developed angular gyrus.

The anatomical relations, therefore, of the upper temporo-sphenoidal convolution on the left were the same as those of the middle temporo-sphenoidal on the right, with the exception that owing to the absence of the angular gyrus the offset from the superior parietal lobule was directly continuous with the temporo-sphenoidal convolution, instead of joining the angular gyrus as it did in the opposite hemisphere. On both sides the uncinate convolution, together with its terminal hook, were well developed, but they were somewhat larger on the left than on the right. On the right the portion of the temporo-sphenoidal lobe lying between the uncinate convolution on the under surface and the superior temporo-sphenoidal on the outer surface of the brain, was divided more or less clearly into the three convolutions ordinarily described, namely, the middle and inferior temporo-sphenoidal, and the inferior occipito-temporal, which is continuous with the latter. On the left the uncinate and inferior occipito-temporal were clearly defined, but the middle and inferior temporo-sphenoidal were fused into one convolution, which was smaller than those which it represented on the opposite side. In fact, the tip of the temporo-sphenoidal lobe on the right projected one inch in advance of its fellow on the left, and was formed by the middle temporo-sphenoidal convolution, which wound round the curved and prominent extremity of the inferior occipito-temporal and projected three quarters of an inch beyond the anterior extremity of the uncinate gyrus. On the left the tip of this lobe was formed by the uncinate convolution and by the extremity of the middle temporo-sphenoidal. The latter, however, did not project, as it did on the right, but was level with the uncinate gyrus. The fissure of Sylvius gaped much more on the left than it did on the right, so that there the island of Reil lay somewhat exposed to view.

The absence of the parts mentioned, viz. the angular

gyrus and superior temporo-sphenoidal convolution, was also indicated by measurements. Thus the fissure of Rolando on each side met the great longitudinal fissure very nearly at the same point, but the right slanted forward much more than the left, the difference being most observable in its lower half. On measuring the distance of the inferior extremity of the fissure of Rolando, where it approached the fissure of Sylvius, from the tip of the frontal lobe, it was found to be on the right three and a half inches, on the left four inches. And the distance of the same point from the tip of the occipital lobe was on the right four and a half inches, on the left four inches. In other words, the lower extremity of the fissure of Rolando was half an inch further back on the left than on the right. This was due to the absence of the angular convolution.

The measurement from the great longitudinal fissure over the angular gyrus and temporo-sphenoidal lobe to the crus cerebri was on the right six and a quarter inches, on the left five and a quarter inches, being a difference of one inch in girth and lateral prominence in favour of the right hemisphere. This was mainly due to the absence of the angular and superior temporo-sphenoidal convolutions on the left.

Thus the measurements bore out the conclusions drawn from a comparison of the anatomical details of the two hemispheres, viz. that the left hemisphere was generally smaller than the right, and that this inequality was due to the reduced size of the individual convolutions, which were otherwise similar on the two sides; but that there was one exception, and that was that the angular and superior temporo-sphenoidal convolutions were absent in the left. Moreover, the middle and inferior temporo-sphenoidal convolutions were fused together on the left, and their place occupied by a single convolution, which was considerably smaller than those which it represented on the opposite hemisphere. Of the other cerebral lobes on the left the parietal appeared to be the most markedly

reduced in size, and to be comparatively smaller than either the frontal or occipital.

When looked at in connection with the condition of the several convolutions already described, the other parts of the brain presented peculiarities, which were very striking and of unusual interest.

The left optic thalamus was hardly more than one half the size of the right, and though on a level with the latter anteriorly it stopped short of it by at least one quarter of an inch posteriorly.

The corpora geniculata, which were well developed on the right, were exceedingly small on the left; and the left optic tract was not much more than one quarter the size of its fellow of the opposite side. The left optic nerve was decidedly larger than the right, but the disproportion between them was not nearly so great as between the two optic tracts, but was about as five to four.

The corpora quadrigemina were well developed and equal on the two sides, but the brachia on the left were much smaller than on the right.

The left corpus albicans was, if anything, slightly larger than the right. The two olfactory nerves presented no difference in size, nor was there any marked inequality between any of the other cranial nerves on the two sides, though those on the left appeared to be a little smaller than those on the right. The anterior and the soft commissure were present, and perhaps a little small. The pineal gland and pituitary body were normal.

The corpus striatum on the left was smaller than that on the right, but the disproportion was not nearly so marked as between the two optic thalami.

The left crus cerebri was decidedly smaller than its fellow, and the left side of the pons Varolii was a little smaller than the right. The disproportion between the two sides of the medulla oblongata was extreme. This appeared to be almost entirely due to the great diminution in size of the left anterior pyramid, which was so small that the olivary body stood boldly out and came to within

one sixteenth of an inch of the anterior median fissure; while the right olivary body was separated from the latter by at least one quarter of an inch. In other words, the left anterior pyramid was about one fourth of the size of the right. The two halves of the cerebellum and their crura appeared to be quite equal in size and similar in every respect.

Both the history of the patient and the anatomical peculiarities of the brain show that we must consider the case as one of malformation of one portion of the left hemisphere and arrest of development of the remainder.

The most striking characteristics of the specimen are:

(1) The general arrest of development of the left hemisphere.

(2) The small size of the corresponding crus cerebri and anterior pyramid.

(3) The absence of the angular gyrus and superior temporo-sphenoidal convolution, together with the fusion of some of the other convolutions of the left temporo-sphenoidal lobe.

(4) The small size of the optic tract, optic thalamus, and corpora geniculata on the same side.

Considering the knowledge we now possess from observations conducted both ante-mortem and post-mortem, it can hardly be doubted that the condition of the anterior pyramids or motor strands must be looked at in connection with the arrest of development of the central convolutions, viz. the two ascending convolutions and their expansions near the great longitudinal fissure. It must be accepted as proved not only that the latter are in some special way connected with the motor impulses transmitted from the cortex to the various voluntary muscles of the body, but also that when their influence is wholly or partially abolished by disease, the motor strands passing along the anterior pyramids gradually undergo atrophy and degeneration, and stiffness and contraction of the limbs result. But more than this it has been shown by several recorded cases that the same atrophic condition of the motor strands

is present, and the same condition of limbs, when the cortical motor areæ are congenitally malformed. Hence it seems to us that we are justified in saying that the arrested development of the ascending central convolutions explains that of the motor tract.

With regard to the frontal lobes no observations drawn either from experiment or disease have succeeded in definitely localising any specific functions in them; nor have we any knowledge of such intimate connection of any of the cerebral nerves with them as would lead us to expect that marked atrophy or degeneration of these nerves would result from their deficiency.

Some physiologists have concluded from their experiments that the occipital lobe contains visual centres, and of late Ferrier himself has inclined to this view. The present specimen tells neither for nor against such a theory. For the slight reduction in the size of this lobe cannot be made responsible for the extreme atrophy of the optic tract; though it is possible that the remnant of the latter which still exists may owe its survival to the presence of the occipital lobe.

The case is different, however, with the angular gyrus and temporo-sphenoidal lobe. Ferrier's experiments have raised a very strong presumption in favour of the theory that the angular gyrus has some special function in relation with sight, some intimate connection with the optic tract. And we hold that the specimen under consideration affords anatomical confirmation of this view. Setting aside the central convolutions and the degenerate motor strands in relation with them, the rest of the left hemisphere, with the striking exception already mentioned, presents only such general slight diminution in size as will explain any inequality which may exist between the nerves proceeding from the two sides. But the angular gyrus and temporo-sphenoidal lobe on the one hand, and the optic tract, optic thalamus, and corpora geniculata on the other, are so exceptionally malformed and deficient, that some interdependence between them is at once

suggested. And although from a consideration of this single anatomical specimen we are hardly justified in making any more precise assertion, still we can scarcely avoid looking upon it as confirmative of those physiological experiments which point to the angular gyrus as in some way bound up with the function of vision.

The opinions of physiologists are still far from unanimous respecting the functions of the optic thalami, nor is it universally allowed that they have any special relation to vision. The great reduction in size of the left optic thalamus in this case, when looked at in conjunction with the still greater diminution of the optic tract must, we think, be taken as striking evidence in support of the doctrine that this large basal ganglion is directly concerned in sight, although this may be far from its only function. The same may be said with regard to the corpora geniculata, about the connection of which with the optic tract there is much less difference of opinion.

The fact that the corpora quadrigemina are well developed and equal on the two sides is of considerable importance, as it indicates that these bodies are not in the direct line of fire, so to say, between the retina and the cerebral cortex. It does not, however, prove that they have no connection with sight. On the contrary, as is widely believed, they may be in some way related to the co-ordination of the ocular muscles, which is necessary for normal vision. And in this patient's case every observer has asserted that the eyes appeared to be healthy and similar in their movements, a fact which would be in accordance with this view of the function of the corpora quadrigemina.

Another point of interest in the case is that it supports the opinion now generally held, that a complete decussation of the optic nerves does not take place in the chiasma, for the left optic tract is very much smaller than the right, whereas the right optic nerve is only slightly smaller than the left. This could not have been the case had complete decussation occurred in the chiasma, for then

the same relative proportion should hold between the two optic tracts and the two optic nerves. But this is not so, and the actual condition of things can only be accounted for on the supposition that to the fibres of the right optic nerve, which come from the left or atrophied optic tract, are added some of the fibres of the right optic tract, which consequently do not decussate.

With reference to the question of the localisation of other functions, such as hearing, smell, and taste, in the temporo-sphenoidal lobe, this case gives a dubious answer. For although, in spite of the abnormal condition of this lobe, there is no marked inequality between the nerves connected with these senses on the two sides, still it is uncertain how far the normal temporo-sphenoidal convolutions are represented in this malformed specimen. Hence it is not legitimate to conclude from the fact the nerves of special sense in question are not reduced in size in the same proportion as the temporo-sphenoidal lobe, that this part of the hemisphere is not in direct functional connection with them. The brain, which has been described, yields no decisive information on this point, but we hold that it does give strong support to the views held by Ferrier as to the localisation of at least a part of the visual centres in the angular gyrus.

DESCRIPTION OF PLATES X AND XI.

Asymmetry of the Brain (SEYMOUR J. SHARKEY, M.B.).

PLATE X.

FIG. 1. Base of Brain.
2. Vertex.

PLATE XI.

FIG. 1. Right Hemisphere (normal).
2. Left Hemisphere.

A. Ascending frontal convolution.
B. Ascending parietal convolution.
C. Supra-marginal convolution.
D. Inferior border of the parietal lobule.
E. Third annectent convolution.
F. Middle temporo-sphenoidal convolution.
G. Convolution formed by offsets of the parietal lobule and second annectent gyrus on the left, and by these together with a branch from the angular gyrus on the right, and running into the middle temporo-sphenoidal convolution.
H. Island of Reil.
K. Angular gyrus.
L. Superior temporo-sphenoidal convolution.

Plate X.

Fig. 1.

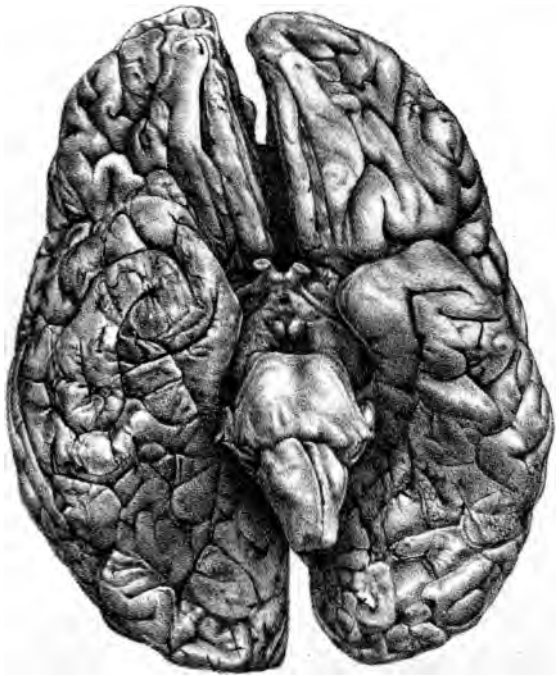
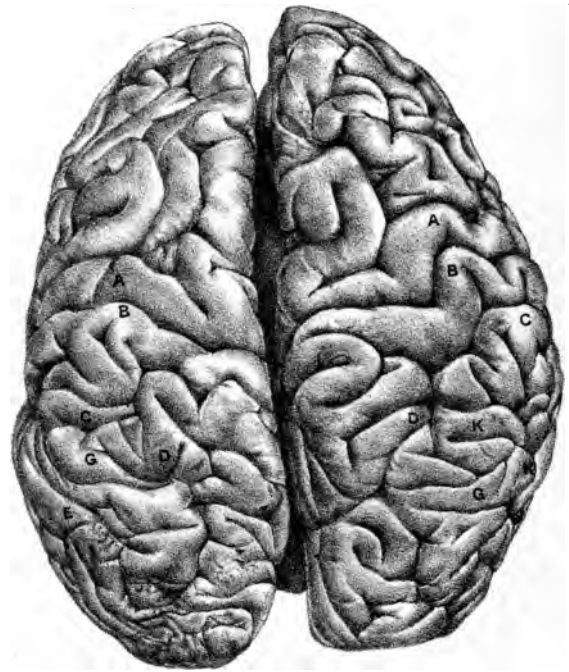


Fig. 2.



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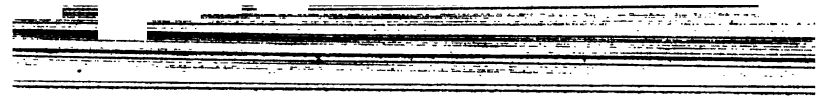

Fig. 1.

Fig. 2.

R. Mintern, lith.

From Photographs by C. West.

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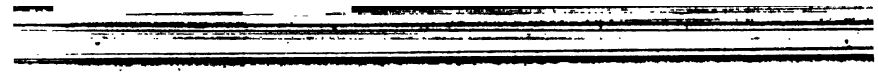


A CASE

OF

EXCISION OF AN ENLARGED CANCEROUS KIDNEY.

BY

SIR SPENCER WELLS, BART.,

SURGEON TO THE QUEEN'S HOUSEHOLD; LATE PRESIDENT OF THE ROYAL COLLEGE OF SURGEONS.

(Received May 8th—Read May 22nd, 1883.)

ON the 9th of December, 1882, in consultation with Mr. Hewer, of Highbury, I saw a German gentleman, æt. 58, who was suffering from the effects of repeated attacks of hæmaturia, supposed to depend upon an enlarged left kidney. The amount of blood lost had seriously affected the general health, and on different occasions it had been with difficulty that the bladder had been cleared from clots. There was no evidence of any disease of the bladder nor of the right kidney. The left kidney appeared to be about four inches in breadth and six inches from above downwards; extending from the iliac fossa upwards under the left false ribs, and centrally nearly to the umbilicus. It was slightly mobile, and could be pushed to the right a little beyond the umbilicus. Feeling that

the surgical difficulties in removing the kidney would not be great, and that the patient was not likely to live long if it were not removed, I advised early operation, and was supported by Mr. Hewer.

The patient took time to consider the question, and consulted his old friend, Dr. Herman Weber, who informed me that, twenty-five or thirty years ago, and until 1871, he had been subject to so-called "bilious attacks" with violent vomiting. In 1871 he had rather extensive pneumonia on the right side, after which he remained free from the attacks of vomiting for many years. Dr. Weber added: "In December, 1880, I saw him again, for the first time after many years. Then he had observed the moveable tumour which had first come to his notice after the last severe attack of vomiting a year before this visit to me.

"In August, 1881, Mr. K— was at Margate, and one day the urine was bloody. On the following day he came up to me, when I found the urine quite free from blood and albumen, perfectly clear, and normal. The first severe bleeding occurred in May, 1882, when I had not seen him for eight months. It ceased completely under the administration, by Mr. Hewer, of turpentine in capsules. In July and August he went to Germany, and was so well that he could climb moderate mountains. By that time I had attributed the tumour to the kidney, and feared that probably it was malignant."

Three days after my first visit Mr. Hewer wrote to me: "12th December, 1882, the patient is losing more blood and consents to the operation, which, I think, should be done with as little delay as possible." Proposing to operate the next afternoon, Mr. Hewer telegraphed to me, "Patient not so well, please make operation, if possible, to-morrow morning." Accordingly, at 9 o'clock on the morning of the 13th of December—Dr. Herman Weber and Mr. Hewer being present—Dr. Day administered methylene, and I was assisted by Mr. Meredith and Mr. Hewer's son, house-surgeon of St. Bartholomew's. Phenol spray was used and the usual antiseptic precautions were strictly observed.

I made an incision parallel with, and about two inches to the left of the linea alba, extending from about three inches above to three or four inches below the level of the umbilicus. I meant to carry the incision along the outer border of the left rectus, but the muscle was spread out and some of its fibres were divided or separated all along the incision. Several small arteries were tied, and the peritoneal coat of the anterior abdominal wall was divided to the extent of six inches. Intestines were pushed aside, kept back by carbolised sponges, and the peritoneal covering of the kidney was divided to the same extent as the incision in the abdominal wall. Several large veins, which were divided with the peritoneal covering of the kidney, were tied. The kidney was then separated from its loose attachments and drawn out. The ureter was first temporarily secured by two pressure forceps and divided between them. I meant to have tied the renal artery and vein before separating the kidney, but I could not feel the artery. I therefore compressed the connecting tissue with forceps, cut away the kidney, then transfixed with a double silk ligature behind the forceps, and tied in two parts. Loose portions of the peritoneal covering of the kidney were cut away and several ligatures were applied to small vessels. The incision in the abdominal wall was united by silk sutures.

Not much blood was lost during the operation, although it occupied nearly one hour and a half from the commencement of inhalation until the patient was in bed. Much time was lost in tying small vessels in the abdominal wall before opening the peritoneum, and in securing vessels in the divided coverings of the kidney; and, during the closing of the wound, there was unusual difficulty in keeping back the intestines free from the sutures.

The kidney is now in the Museum of the Royal College of Surgeons, and Mr. Eve's report on the specimen is as follows:

"The kidney is six and a half inches long and four wide. Its surface is largely nodulated or bossed; but

the disease had not penetrated the capsule. The section of the tumour is white, soft, and marked by bands of fibrous tissue, which give it a lobulated appearance. Portions of unaffected kidney substance are situated at the upper and back parts of the section. Neither the ureter nor pelvis were compressed. Microscopically the morbid growth was a soft cancer, consisting of alveoli filled with small spheroidal epithelium."

For three days after the operation the patient went on so well that I was hopeful of recovery. He suffered very little from pain or sickness, but the urine never became free from blood. On the fourth day the temperature rose to 102°, and the pulse became rapid and feeble. He was much worse at night, and died on the morning of the fifth day. The temperature just before death was 103°, and the last urine passed contained blood. No post-mortem examination of the body was permitted. This I very much regret, as I cannot explain the continuance of blood in the urine in the absence of any proof of disease of the right kidney or of the bladder, nor can I offer any reliable reason of the precise cause of death. There was no evidence of more than slight peritonitis, nor of septicæmia. Mr. Hewer thought the enfeebled state of the patient from continued loss of blood had a great deal to do with the result, and that the chances of success would have been much greater if the operation had been done six months earlier.

If I should again be called upon to excise a kidney, I shall be more careful in bringing together not only the divided peritoneum of the anterior abdominal wall, but also the divided peritoneal covering of the kidney. In this case I was content with letting the two edges fall together; and it is probable that blood or serum, exuding from the tissues behind the peritoneum, passed into the peritoneal cavity, or that some portion of intestine became adherent there. Both these evils would be prevented by a few sutures. I have not seen this detail in the operative procedure described in cases of nephrectomy hitherto

recorded; and the hope that it may be tried, and may assist in diminishing the mortality of the operation, is my best reason for bringing this case before the Society. If this excuse be not accepted as valid, I would add that when a comparatively new operation is on its trial, it is of great importance that every case, especially the unsuccessful cases, should be made known, and that the lessons they teach should not be lost.

A CASE

OF

SUPPURATING TRAUMATIC HÆMATOMA

CONNECTED WITH THE LEFT KIDNEY,

TREATED BY

PUNCTURE AND ANTISEPTIC DRAINAGE.

BY

JOHN MARSHALL, F.R.S.,

PROFESSOR OF SURGERY IN UNIVERSITY COLLEGE, AND SENIOR SURGEON TO UNIVERSITY COLLEGE HOSPITAL; PRESIDENT OF THE SOCIETY.

Received May 22nd—Read May 22nd, 1883.

In March, 1882, A. M—, a girl, æt. 13, was knocked down by a spring cart, one of the wheels being said to have passed over her abdomen; she was then unable to stand, and complained of severe pain in the left flank, passing inwards towards the umbilicus, and also affecting the lumbar spine. She was carried home and remained in bed a month, during which period the pain became less, though it did not disappear entirely and was increased by movement. At the end of a month the patient left her bed, but was only able during the next month to walk about the house. The pain continued nearly as severe

until August, five months after the accident, when it diminished, so as to allow of the child going to school. In December, that is nine months after the receipt of the injury, the pain returned in all its former severity, and the patient was admitted into the University College Hospital.

There was no history of hæmaturia or other urinary trouble at any time.

On examination (Dec. 22nd), the patient was found lying on her right side with her legs drawn up, looking pale and anæmic; she had a suffering and very anxious expression, and was considerably emaciated; she complained of great pain in the left lumbar region extending inwards to the umbilicus, and made worse by movement. The abdomen was distended, especially on the left side, by a swelling, which occupied the left lumbar region and left half of the abdomen. The abdominal muscles were very tense and resisted palpation; the tumour was so excessively tender that the patient could not bear to be examined. The tongue was slightly furred; the temp. was 103° F.; the pulse 128, rather compressible; the urine scanty and high-colored.

On the third day after her admission (Dec. 26th), the temperature having then fallen to 99·6°, the patient was put under chloroform and examined, when the tumour was found to completely fill the left lumbar region, extending *downwards* to the anterior superior iliac spine, *inwards* 1½ inches to the right of the middle line level with the umbilicus, and *upwards* to the margin of the thorax. There was no interval behind, between the tumour and the mass of the erector spinæ, and no connection with the pelvis. Distinct fluctuation was obtainable across the tumour, which was dull on percussion, a band of resonance, however, crossing it in front and above.

An aspirator needle being introduced into the most prominent part of the tumour, a little to the left of the umbilicus, five fluid-ounces of a yellow-brown odourless fluid were drawn off. When heated this liquid emitted a

strong urinous odour, and yielded a heavy flocculent albuminous precipitate. It was found to contain ·55 per cent of urea. Pettenkofer's test gave no bile-reaction.

Great relief followed the aspiration; there was scarcely any pain; the temperature was normal; there was no urinary trouble. Eight days later, however, the pain returned; the temperature rose to 103·2°; the pulse, which was compressible, to 124; and the respirations to 40. There was great tenderness over the tumour, which was more tense than before. Accordingly on January 12th, 1883, the patient being under ether, a large trocar with cannula, one-third of an inch in diameter, was introduced at the site of the former puncture, and thirty-six fluid-ounces of a chocolate-coloured fluid, similar in character to that before obtained, were drawn off. This cannula was left in, and a small drainage-tube, rather more than six inches long, was introduced through it. The whole operation was conducted under carbolic spray, and a dressing of carbolic gauze and waterproof was applied. On the following day the temperature fell to the normal standard, and the pain almost entirely ceased. There ensued a profuse discharge of a brownish-red colour, having the appearance of broken down blood-clot. During the next seven days, the wound being dressed antiseptically at intervals, the discharge consisted of pus mixed with a brownish-red fluid. The cannula was then removed and the drainage tube only left in. On the tenth day, while the dressing was being renewed, there was a gush of about two fluid-ounces of clear fluid resembling urine, followed by a small quantity of pus. No more clear fluid subsequently escaped from the wound, and from this time the recovery was slow but uninterrupted. The patient was discharged cured on April 16th, 1883, about thirteen months after the accident, and five months after the consecutive suppuration had led to her admission into the Hospital. The patient appeared and felt well; the area of dulness was exceedingly small; the wound was quite superficial and almost closed.

From the time of the operation onwards the patient passed from twenty to forty ounces of urine daily, the urea varying from 2·4 to 3·6 per cent.

The diagnosis made in this case was that of a large, tense, fluctuating tumour in close relation with the left kidney, which tumour, from the history of the accident, and of the severe consecutive local and general symptoms, was probably a *traumatic hæmatoma*, which had undergone incomplete absorption, and had ultimately *suppurated*. The alternative suggestions of a cyst, or of a soft sarcoma of the kidney were considered, but for obvious reasons were thought to be less tenable.

The sanguineo-purulent character of the chocolate-coloured fluid drawn off from the tumour, confirmed the notion of its being a suppurating blood-tumour; whilst the urinous odour detected on boiling that fluid, the fact of its containing ·55 per cent. of urea, and the curious gush of clear fluid which escaped during one of the dressings, pointed to a superficial laceration of the kidney itself, unless indeed the injury gave rise to a sub-capsular hæmorrhage, or again unless the bleeding had occurred outside the kidney and the urea had passed into the sac of the tumour by a process of osmosis.

The complete recovery of the patient, after a simple, but wide puncture and persevering antiseptic drainage, gives special interest to this case.

ON THE BACILLUS OF LEPROSY.[1]

BY

GEORGE THIN, M.D.

(Received April 10th—Read June 12th, 1883.)

THE pathology of tubercular leprosy (Elephantiasis Græcorum) is intimately associated with two discoveries. The first series of observations, which defined the group of diseases amongst which leprosy is now classed, were chiefly concerned with the cell-growth that is invariably found in the tubercles. The second series, which were initiated by the investigations of Armauer Hansen in Norway, are concerned with an organism, presumably foreign to the human body, which is found in the cells of leprous tissues—the so-called *Bacillus lepræ*.

With the cell-growth of leprosy I shall not attempt to deal in this paper. It has been frequently and well described in the numerous memoirs, monographs, and text-books of pathology that have been published during the last decennium. Comparatively little has been added to our knowledge of the ordinary pathological histology of

[1] The research which is reported on in this paper forms part of a series of investigations on the parasites of the human skin, towards which a grant has been made by the Scientific Grants Committee of the British Medical Association.

the disease since the description given by Virchow in the second volume of his work on 'Tumours,' published nearly twenty years ago. It is well known that that eminent pathologist grouped together syphilis, lupus, and leprosy, as forming a class of granulation tumours; these three diseases having in common, destruction of tissue and the formation of groups of cells which bear a resemblance more or less marked to granulation cells.

Of the two appearances, the destruction of fibrous tissue and the grouping of cells, the greatest weight has been assigned to the latter, the cell of leprous tissue being supposed to be intimately associated with the development of the disease.

The first notice of the bacillus of leprosy is contained in a report made to the Medical Society of Christiania in 1874 by Hansen. In his paper on the subject in the 'Quarterly Journal of Microscopical Science,' [1] this report is referred to as containing the statement that he had "often, indeed generally, found, when seeking for them in the leprous tubercles, small rod-shaped bodies in the cells of the swelling."

The date of this report fixes that of the first publication of the discovery, and assigns it indisputably to Hansen.

With commendable caution Hansen was slow to assign too much importance to the appearances which he had observed, and in his paper in the 'Quarterly Journal of Microscopical Science' (published simultaneously in French and German), we still find him hesitating to regard the bacilli as the cause of leprosy. Having failed to inoculate rabbits by introducing portions of the leprous growths under the skin of the animals, he considered the failure was adverse to the supposition that the bacteria are the real virus. His paper is illustrated by a plate giving drawings of the organisms.

The existence of bacilli in lepra-cells has been confirmed by Neisser, Cornil and Suchard, Koebner, and others. It only remains now to extend the investigation,

[1] New series, vol. xx, 1880.

so far as the presence of these organisms is concerned, to a larger number of cases of leprosy, taken from widely separated countries. In considering the question of the connection of the disease with the presence of the parasite, it becomes important to know whether the bacilli are found in all cases and in patients in all parts of the world, and it is on these points that the evidence I now place before the Society chiefly bears.

In the summer of 1882 my friend Dr. Manson, of Amoy, brought me leprous tissue from China, from three cases which had been under his observation.

Case 1.—The leprosy had been known to exist for three to four years, and was a case of mixed anæsthetic and tubercular leprosy. There were tubercles on the face, anæsthesia, and muscular atrophy of the hands and arms, and swelling, bronzing, and anæsthesia of the feet and legs. I examined a tubercle which was excised from the face.

Case 2.—The leprosy had been known to exist for five to six years. There was leontiasis of the face, anæsthesia, bronzing, and swelling of the legs, feet, arms, and hands.

Case 3.—The leprosy, which was of the anæsthetic and annular type, had existed for three years, the patient being a male, æt. 24. A small, round, slightly elevated, dusky-red patch on the abdomen, which had existed for only nineteen days, was excised in its entirety.

Dr. Manson placed the excised portions of tissue in alcohol immediately after he removed them from the living patients. He brought them with him to England and I received them from his own hands. From the time that they were removed from the patients until the time when their preparation for microscopic observation was completed, the possibility of the development of organisms in the tissues was excluded.

I was also favoured with leprous tissue from the West Indies by Dr. John D. Hillis, F.R.C.S., medical superintendent of the General Leper Asylum, British Guiana, the author of an able and instructive work on West Indian Leprosy. Portions of skin with the tubercle were excised

from the face and right pinna of a black native, æt. 27, affected four years with pure tuberculated lepra. The tissue was taken from the living subject, placed immediately in alcohol, and then in absolute alcohol. They were received by me in alcohol in a well closed bottle.

I shall pass very briefly over the methods by which I endeavoured to show the presence of bacilli in these leprous tissues. They consisted simply in attempts to follow closely the methods which have been published by Koch and Ehrlich. These attempts, for reasons which I can hardly understand, were at first not very successful, then came a stage during which I found it comparatively easy to show the bacilli but found it impracticable to make permanent preparations. Latterly, and for some months past, I have found it easy to show the bacilli and not difficult to make permanent preparations. These improvements in preparation were partly due to greater experience in manipulation, and partly to modifications in the method of operating. The *technique* of this kind of work is, according to my experience, still very imperfectly developed. Whilst the demonstration of bacteria in fluids, as the bacilli of tubercle in sputum for example, is so easy that any intelligent person can be taught to succeed in it in a very short time, the demonstration of organisms in tissues preserved in alcohol, is in many instances by no means easy and in many cases fails. A certain method, applicable to all cases in which organisms are present in solid tissues, and of easy application, is yet to be discovered. As my own efforts in this direction are still progressive in their results, I think it better to reserve anything I may have to contribute on this part of the subject for future publication. In the meantime I may state that the demonstration of the bacilli of leprosy by staining with gentian violet and methylaniline violet, and fuchsine as recommended by Professor Ehrlich for tubercle bacilli, will not be found difficult by anyone who is familiar with work of this kind.

The bacilli described by Hansen were found in the skin

of the three Chinese lepers and in the skin sent me from the West Indies. They were also found in the epiglottis of a leper who died in Australia. This patient was born in New South Wales and was the son of an English father and an Irish mother. I am indebted to my friend Dr. Maclaurin, of Sydney, for sending me the larynx, and to Dr. Cox, of Sydney, for notes of the case.

Of the three portions of skin from three different patients brought me by Dr. Manson, I possess permanent preparations of one only. But bacilli were also observed in the skin from the other two cases.

My description is based on the case of Chinese leprosy, of which I have been able to preserve specimens, and on the specimens of Demerara leprosy.

When a vertical section through the skin of a leprous nodule, prepared for the demonstration of bacilli, is bleached to a certain degree, all the tissues of the cutis become colourless, and the leper cells alone are stained. These stand out boldy, deeply stained with methylaniline violet, if that dye has been used, whilst the surrounding connective tissue does not show a trace of staining. Such a section is suited for observing the size and form of the leper cells. The whole cell is coloured uniformly and no nucleus is visible. If the connective tissue amidst which the cells lie is carefully examined, no bacilli are found in the bundles or in the interfascicular spaces (so much at least has been the result of a careful and repeated examination of my sections prepared from these cases).

If the bleaching process is carried a degree further, bacilli are seen in some of the cells—those in which the decolorisation is most advanced—whilst the other cells are still seen as deeply-stained masses. The longer the bleaching is continued the greater the number of cells which are seen to contain bacilli, until a point is reached at which the bacilli themselves become faint.

In a section in which the bleaching has so far advanced as to show many bacilli, the organisms may be seen grouped within the contour of a lepra cell, or they may

be seen singly or in small numbers apparently lying free in the tissues. I am disposed to believe that the latter appearance is deceptive. In the more deeply-stained sections no individual bacilli are observed, cells alone retaining the dye. Some of these cells are indeed very small, many of them, especially in parts in which the leprous infiltration is beginning, being as small as white blood-corpuscles. It is probably due to the small number of bacilli contained in these minute cells that the appearance of apparently isolated bacilli in more completely bleached sections is due.

In the parts of the skin not affected with the leprous infiltration no bacilli were observed.

During the preparation and study of a large number of sections I have come on two appearances (both hitherto, so far as I am aware, undescribed) of the greatest significance.

It has been remarked by more than one observer that when the epidermis is entire in non-ulcerated leprous tubercles no bacilli are found in the epidermis. Cornil and Suchard,[1] for example, use the following words:—"A remarkable peculiarity which we have observed in all our preparations is that the different layers of the epidermis do not contain any microbe, the epidermic covering forming a varnish impermeable to the special parasite of leprosy. We shall return to this point in studying the conditions of this malady compared with the contagion of other cutaneous affections;" and, again, they state further, "that the epidermic layers although thinned are free from parasites. This epidermic layer opposes itself, so long as it is preserved, to the external diffusion of the parasite, and forms a barrier to it which renders contagion very difficult. The site of the parasite is deep. The contrary is the case in most parasitic affections of the skin which are specially contagious, such as eruptive fevers—smallpox, for example, and erysipelas."

[1] "Note sur le Siége des Parasites de la Lèpre," 'Annales de Dermatologie, &c.,' 2me série, vol. ii, p. 655.

I have found in one of my sections bacilli in the epidermis. Whilst the tissue was deeply stained the appearance was observed of small cells, which retained the aniline colour, being scattered through the epidermis at different points. After the section had remained some time in oil of cloves I was able to observe that the colour of these cells was due to their containing bacilli. The size of the cells was that of white blood-corpuscles. I made a camera drawing of the preparation, and Mr. Thurston, to whom I am much indebted for the care he has taken in preparing the plates which accompany this paper, made an independent camera drawing of the same preparation. The position and number of the bacilli in the two drawings corresponded. A reference to the drawing on the plate renders it unnecessary for me to describe them further. The epidermis surrounding these cells was quite uncoloured, of normal thickness and texture. The leprous infiltration of the section was chiefly in the deeper part of the corium, the papillary layers being intact, and containing very few bacilli.

I believe that the bacilli in the epidermis in this section were contained in white blood-corpuscles which had passed into the epidermis, but I am prepared to admit that this view is open to discussion.[1]

The other appearance was again found in a solitary instance. In this section, also, the leprous infiltration was in the thicker and deeper layer of the cutis, the papillary layer being entire and containing very few bacilli. In the papillary layer, midway between the epidermis and the leprous infiltration, a lymphatic vessel was seen lying parallel to the epidermis, and containing two lymph-corpuscles in its lumen. In both of the corpuscles bacilli could be seen, but in one of them more distinctly and in greater number than in the other. Part of the lymphatic vessel, containing the corpuscle in which the

[1] Although since the above was written I have examined many sections in which the staining was perfect, I have not again met with a similar appearance (July 18th).

bacilli were most numerous, is shown in the drawing (Pl. XII, fig. 1).

The accuracy of the observation was again tested by Mr. Thurston and myself making independent camera drawings which almost exactly corresponded. The only point in which my observation differed from that of Mr. Thurston is that, in Mr. Thurston's drawing a bacillus is lying free in the lumen of the vessel close to the corpuscle; I included this bacillus in the corpuscle. The difference probably depends on the respective focuses used in making the drawings.

This seems the place for me to call attention to three facts, and to the theory which I believe they support.

It has been frequently observed that the smaller cells of leprous infiltration resemble white blood-corpuscles, and in their size and appearance I have found no difference between them and the ordinary small cell-infiltration with which we are familiar in many pathological conditions of the skin.

It has been stated above that in the epidermis, cells similar in size to white blood-corpuscles, have been found containing bacilli, and in the lymphatic vessel a lymph-corpuscle has also been found containing the organisms.

To my mind these appearances suggest the theory that the lymph-corpuscles are one of the media by which the bacilli are carried from one part of the body to another. The fact that the infected lymph-corpuscle has been found in a lymphatic vessel, although as yet, so far as I know, unique, enables us to understand how the disease is carried from the skin to internal organs. Although the fact in support of this theory is yet isolated, it will be granted that it renders it a highly plausible one.

The groups of bacilli found on the borders of the lymphatic vessel in the section which I have described, are in all probability due to the fact that lymph-corpuscles had passed from the vessel into the adjacent connective tissue. In any case it is clear that they had an intimate relation to

the vessel, because the surrounding connective tissue and the papillary layer were free from organisms.

Some experiments were made with the view of ascertaining whether it is possible to show in the healthy skin organisms similar to those found in leprosy. With this view, sections of healthy skin from the female breast and the leg of a man, and sections from an epitheliomatous tumour of the vulva, and from the skin of several patients who had died of scarlet fever, were submitted to the same process by which the bacilli of leprosy were demonstrated, and in no instance were any such organisms observed. Further, in the leprous tubercle itself the connective tissue of the healthy skin, adjacent to the infiltration, showed no bacilli. We are therefore warranted in concluding that the organisms found in the cells of leprosy have an intimate connection with the disease.

During the progress of this investigation, whilst already engaged in preparing this paper, I received from Sydney the larynx of a man who died of leprosy in the Liverpool Asylum of New South Wales. I have received a detailed account of the case, drawn up by Dr. Cox, of Sydney. The case will be published hereafter when my examination of the larynx is more complete. At present I am only desirous of placing on record the size and appearance of the bacilli in a case of leprosy having its origin in Australia. The patient had been affected with genuine tubercular leprosy for six years at the time Dr. Cox drew up the notes. An addendum to his report states that the patient died two months afterwards from suffocation, a result which will astonish no one who examines the larynx which I have received. Sections have been prepared from a small portion of the greatly thickened epiglottis, and they show bacilli of the same appearance as those found in the cases which I have already described.

The evidence of the association of a bacillus with the leprous infiltration at present stands thus :—It has been found in Norwegian leprosy by Hansen and other Norwegian observers; it has been found by Cornil and

Suchard in specimens taken from the leper asylum of Grenada, and from a patient under treatment in Paris; it has been found by Koebner in a patient who had contracted the disease in South America; by Majocchi and Pellizzari in two patients from the Island of Elba; and I have now found it in skin excised from patients in China, in the West Indies, and in the larynx of a leper who was born, and died, in Australia. The intimate association, therefore, of the parasite with the disease seems indisputable.

I am not prepared to lay too much stress on the size, form, or appearance of bacilli as indicating identity of kind, but anyone who compares the drawings that have been published of the lepra bacilli with each other must be struck with the similarity in these respects which the organisms seen by different observers have to each other. The drawings by Hansen,[1] Neisser,[2] Cornil and Suchard,[3] and of Majocchi and Pellizzari[4] all closely resemble each other. The question of size is a somewhat difficult one. Foreign observers are in the habit of indicating size by the numbers of the eye-pieces and objectives which they use in making their drawings. It has been my object in preparing this paper to lay before the Society something which, in England at least, will be considered more definite.

I have been fortunate in securing the assistance of Mr. Edgar Thurston, who is highly competent in the difficult task of drawing minute organisms to scale. The plates which he has prepared show the bacilli in Chinese leprosy, in West Indian leprosy, and in Australian leprosy, as magnified by a fixed number of diameters; and in order to afford standards of comparison, drawings have been made of the bacilli of tubercle, of the bacilli found in sections of a putrid sheep's cornea, and of the bacilli

[1] Loc. cit.

[2] 'Virchow's Archiv,' vol. 84.

[3] Loc. cit.

[4] 'Studii Ematologici nei Leprosi,' Florence, 1882.

which formed a large part of the scum of cold mutton infusion. A comparison of these drawings will show that so far as regards the size of the various bacilli, erroneous statements have been made.

It will be noticed that the bacilli in the epiglottis of the Australian leper, while they are of the same length as the bacilli in the other leper specimens, appear more slender. This is probably due to the fact that the larynx from Australia had been for a considerable time in alcohol before the tissues were examined.

The bacilli are generally found single but occasionally they are found of the double length, the protoplasm of two rods being contained in one sheath, a distinct space being seen between the rods.

The existence of bacilli in the blood of leprous patients has been affirmed and denied. Koebner and Majocchi and Pellizzari state that they have found them in the blood. My own observations on this point are very limited. I have on several occasions examined the blood of two lepers, both of them of English descent, who had contracted the disease in Hindostan. In one of them, an advanced case of the disease, leper cells were observed in the blood freshly drawn from a finger affected with the infiltration. In some of these cells, examined fresh in the serum, appearances were noted which were highly suggestive of bacilli. I should not, however, have felt warranted in affirming their existence had it not been that one of these cells, whilst under observation, was ruptured by the pressure of the cover glass and a number of bacilli were set free, the distinctive form being easily observed. In the other case, a less advanced example of the malady, leper cells were also on several occasions observed in the blood, and on one occasion two rods were observed free in the serum.

Postscript (June 6th, 1883).—Since the above paper was sent to the secretary of the society I have examined, as a member of a committee appointed by the Clinical

Society, tissues excised from leprous tubercles in the arm of a boy who had acquired the disease in Hindostan. Bacilli were found in numbers in the excised skin.

In this case I detected, for the first time, bacilli in the blood-vessels of the tubercles, and I have since found the parasites in blood-vessels in another case.

The bacilli in this East Indian case were of the same size and form, and exhibited the same peculiarities, in regard to the action of aniline dyes, as those found in West Indian and Chinese leprosy.

I have also, since the paper was written, obtained preparations in which bacilli stained by fuchsine were seen to be beaded or filled with spores, thus showing a resemblance in another point to the tubercle bacilli.[1] Indeed, with the discovery of spores in the rods the similarity as regards shape, size, and apparent structure appears complete; a fact that becomes more important when we consider that the bacilli in both diseases have the remarkable quality of retaining the fuchsine stain after the bleaching action of dilute nitric acid has removed it from the surrounding elements and tissues.

In the present state of our knowledge of micro-organisms I deprecate drawing any hasty conclusions from this resemblance, but the fact is worthy of being recorded and of receiving attention.

[1] The demonstration of spores in the rods appears to be entirely a question of definition and illumination. I have fuchsine preparations in which nearly all the bacilli appear beaded.

DESCRIPTION OF THE PLATES XII AND XIII.

The Bacillus of Leprosy (GEORGE THIN, M.D.).

PLATE XII.

Fig. 1.—Vertical section through a tubercle in West Indian leprosy. (The leprous infiltration in the corium, below the part shown in the drawing, was dense.) The drawing shows part of a lymphatic vessel, containing an infected lymph-corpuscle, in the papillary layer of the cutis. *a*, The highly pigmented rete mucosum of the negro skin. (The individual cells of the rete are not drawn.) *b*, The papillary layer of the cutis; *c*, a group of bacilli close to the wall of the lymphatic vessel, the cell in which they were contained (as the author believes) not being visible on account of the extent to which the bleaching has been carried; *d*, a lymph corpuscle containing bacilli, lying free in the lumen of a lymphatic vessel. × 1230 diameters.

Fig. 2.—Vertical section through a tubercle in Chinese leprosy, showing bacilli in the unbroken epidermis. *a*, The horny layer of the epidermis; *b*, group of three bacilli, corresponding to one cell; *c*, deep surface of the epidermis; *d*, papillary layer of the cutis. × 950 diameters.

Fig. 3.—Vertical section through a tubercle, under a low magnifying power in order to show the disposition of the leprous infiltration. *a*, The epidermis; *b*, the papillary layer of the cutis; *c*, a hair; *d*, the leprous infiltration. The cells are seen deeply stained with the aniline dye. × 75 diameters.

PLATE XIII.

Fig. 1.—Showing bacilli in the leprous infiltration of a tubercle in Chinese leprosy. The contours of some of the cells are seen with the bacilli in the cells. Amongst the bacilli which appear to be lying free, the arrangement in groups in most instances indicates the position of cells in which they are contained, the cells being no longer visible. × 1100 diameters.

Fig. 2.—Showing the bacilli in the leprous infiltration in a tubercle in West Indian leprosy. × 1100 diameters.

Fig. 3.—Bacilli from West Indian leprosy, stained with fuchsine, and showing a beaded (spore) appearance. (Compare with Figs. 4 and 5, which show bacilli of tubercle, drawn from a preparation of Mr. Thurston's.)

(Figures 4, 5, 6, and 7 are introduced to afford objects of comparison with the bacilli of leprosy.)

Fig. 4.—Bacilli of tubercle, very highly magnified. × 2350 diameters.

(Figures 5, 6, and 7 are drawn with the same magnifying power as the bacilli of leprosy in Figs. 1 and 2.)

Fig. 5.—Bacilli of tubercle. × 1100 diameters.

Fig. 6.—Bacilli in a section of a putrid sheep's cornea. × 1100 diameters.

Fig. 7.—Bacilli from scum on cold mutton infusion. × 1100 diameters.

Fig. 8.—Bacilli of leprosy in the epiglottis. (Australian case.) × 1100 diameters.

Fig 1.

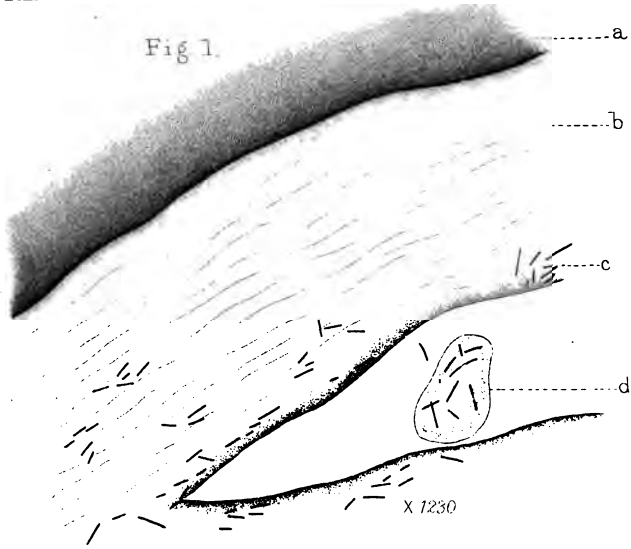


Fig 2.

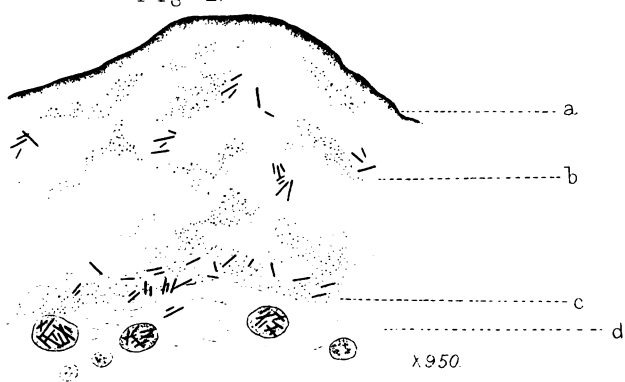
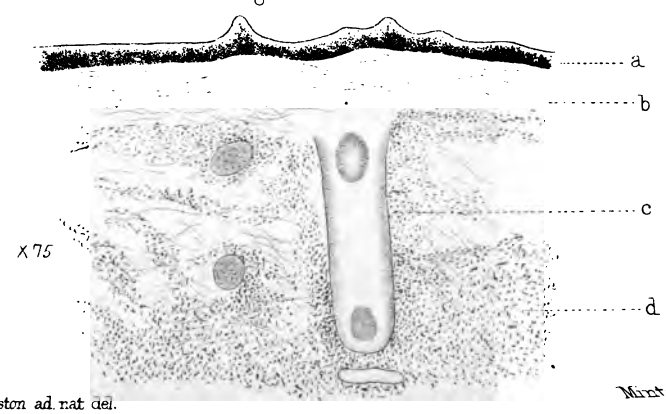


Fig 3.



Edgar Thurston ad. nat. del.

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Fig. 1.

Fig. 2.

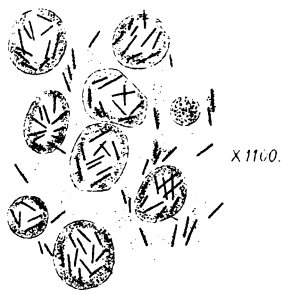


Fig. 3.

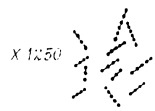


Fig. 8.

Fig. 5.

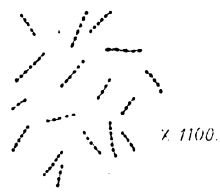


Fig. 4.

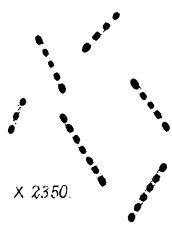


Fig. 7.

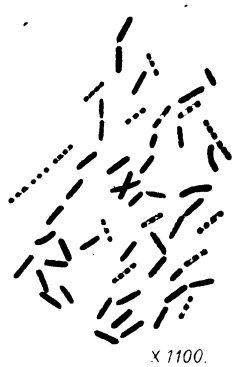
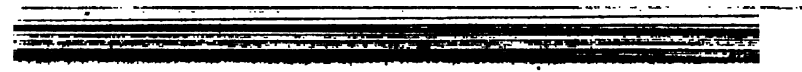


Fig. 6.

Edgar Thurston ad nat. del.

Mintern Bros.



ON

URTICARIA PIGMENTOSA

OR

XANTHELASMOIDEA.

URTICARIA PIGMENTOSA (SANGSTER),
U. PERSTANS PIGMENTOSA (PICK),
XANTHELASMOIDEA (TILBURY FOX).

BY

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(Received May 8th—Read June 12th, 1883.)

THIS rare affection was noticed first by Mr. Nettleship in 1869, but his brief description entirely escaped attention until after Mr. Morrant Baker and Dr. Tilbury Fox had exhibited cases to the Clinical Society in 1875. In the following year (1876) the eruption was met with by Dr. Albert Morrow, of New York, under the name *Erythema tuberculatum with unusual features*. With regard to the exact nature of the affection considerable uncertainty has existed. Mr. Baker, notwithstanding the persistence and inactivity of the eruption in his case, inclined to regard it as *urticarial* in nature, although Dr. Thin, from subsequent microscopical examination of the lesions, concluded that the morbid changes were indistinguishable anatomically from the earlier lesions seen in *lupus*.

In the case of the boy Edward Cornell, shown by Dr.

Tilbury Fox, the eruption was probably more marked and intense than in any other, with the exception perhaps of Dr. Morrow's. The "nodules" resembled those of xanthelasma so closely in appearance, and in the implication of the eyelids, penis, and palate, that Dr. Fox came to the conclusion there was "clearly a deposit of buff-coloured stuff in the skin," and he suggested the term *Xanthelasmoidea* for the reason that, to quote his own words, "acute general xanthelasma would roughly describe the disease in my cases; but if it be thought that the disease is like, yet not the same, as xanthelasma, I should be disposed to term it *Xanthelasmoidea*." Dr. Fox subsequently adhered to the opinion that though there was an urticarial element present the eruption was not urticaria.

My objects in bringing this subject before the Society this evening are:—(1) To present the notes of an additional case; (2) to trace the history of Dr. Tilbury Fox's three original cases down to the present time, *i.e.* over a period of about ten years each; (3) to furnish the results of a microscopical examination of the eruption, as seen in the boy Edward Cornell; and (4) from the examination of the nineteen cases which have so far been recorded, to draw conclusions as to the nature and course of the affection.

The following unpublished case was sent by Dr. Propert to Dr. Tilbury Fox in August, 1877. The boy was then eight months old, and the surface generally was studded more or less with eruption. According to the mother's statement, they noticed on the child at the age of five or six weeks "distinct little blisters," which gave place to brownish-red raised patches. The eruption subsequently appeared as copper-coloured blotches under the skin. The mother thinks they commenced on the head and forehead, and later spread between the shoulders and over the back and trunk generally by the time the child was three months old. At eight months the face was certainly only very slightly involved.

In May, 1878, *i.e.* at sixteen or seventeen months of

age, the surface was thickly sown with slightly raised, congested, coarsely granulated spots and with pigmentary stains, mostly discrete, oval, or rounded, and the size of a split pea, but many larger and irregular from confluence with an ill-defined outline. The flanks were much affected, the abdomen and upper extremities moderately, and the legs increasingly so. Fresh lesions, which had recently appeared on the forehead and face, were more hyperæmic than the older ones, and had distinctly an urticarial element; a little later they become like erythema papulatum only with a deeper, duller red tint. The colour and amount of irritation they gave rise to, however, varied, for when quiescent for some time they acquired a brownish or buff-coloured tint, and were but little itchy, but if irritated—for instance, by rubbing—they get hyperæmic and itchy. When the skin was irritated by washing, &c., white wheals arose. The mother suffers from urticaria, but there is no struma and no phthisis on either side, and the father's family is an exceptionally healthy one. The child under consideration also is fairly healthy, as are his brothers and sister. It may be mentioned, however, that one had spurious jaundice on the third day after birth. In 1880 the eruption was in about the same condition.

With regard to the three cases recorded by Dr. Tilbury Fox in the Clinical Society's 'Transactions' for 1875, I have to state that they have been under observation now respectively for eleven, ten, and nine years. Dr. Brodie kindly informs me that he frequently sees one boy, and the eruption has almost entirely disappeared. A second boy, now nine years old, I saw in March of the present year. He is fairly healthy and well grown, but slightly choreic. On the limbs, trunk, and neck, but especially the legs, are large, irregular, tawny-olive stains, with an ill-defined outline. They cannot be made to assume an urticarial condition by any amount of rubbing. Since 1878 fresh very irritable spots (almost certainly urticaria) have appeared from time to time "when the child was out of sorts," but they have long ceased to leave stains on

their disappearance, and the old raised eruption and the majority of the stains have quite gone.

The third case I show here to-night, and for comparison with the boy's original condition I hand round the water-colour drawing which was executed when he was a small child.

The boy is distinctly more pallid and less well grown than the rest of the family, though on the whole in fair health. In 1878, *i.e.* five years after the first observation, the eruption was slowly declining in intensity, for the lesions were getting smaller and less numerous. The older ones readily assumed the urticarial condition, however, on being rubbed. From that time the eruption evolved less copiously year by year, and many old stains disappeared, so that at the Congress in 1881 the difference in the appearance of the patient was very striking to those who had seen him in 1875. Nevertheless, a wheal-like eruption still continued to appear from time to time, but it was noticed that the majority of them disappeared pretty quickly and failed to leave the stains they formerly did. At the present time nothing but old stains are to be seen, and these evidently are slowly fading. The mother states that the boy often complains of itching and pricking, and when she looks to see the cause, a wheal is observed which comports itself as an ordinary urticaria.

I will now proceed to briefly summarise the results derived from the examination of the records of nineteen cases. Of these fourteen were boys, four girls, and in one the sex was not mentioned. The eruption was noticed in the first six months of life; in three or four instances in the first few days of existence. It is variously described, in the language of the mothers, as commencing with "white bumps," "watery blisters like scalds," dull red patches, pink spots, and white spots surrounded by redness. Ordinary wheals have been stated to co-exist with the special eruption by those who make a distinction between the two. Some have looked upon all these primary eruptions as undoubtedly urticaria, whilst others have hesitated

to pronounce a definite opinion. With our present knowledge it seems impossible to doubt that the eruption is urticaria, at any rate, it has a marked urticarial element in it. The special wheal-like lesions evolve suddenly without prodromata and may be more or less irritable.

It is a marked peculiarity of the eruption that the hyperæmia is more lasting than in an ordinary urticarial wheal, so that the lesions remain for a long time incompletely subsided, that is to say, the effused fluid is more or less absorbed, but still the area remains hyperæmic and somewhat raised, with the skin in wrinkles as if permanently stretched, just as we see it sometimes in œdema of the skin from dropsy. Whilst in this condition the urticarial state is easily lighted up again by rubbing the spot and probably by emotional disturbance. In the more intense conditions the wheal-like lesions become considerably raised as if there was a marked cellular or other infiltration or some organisation of new tissue; they acquire a firmer feel than normal skin, and the surface gets corrugated, or more or less granular, probably from the fact of the follicles or the attachments of strands of fibrous tissue bending down the stretched surface here and there. Dr. Morrow marked such nodules with indelible ink and found that they remained stationary for one to three weeks and then subsided into pigmentary macules. This brings us to speak of the second peculiarity, viz. that in both the phases just described there is added a yellowish or brownish pigmentation irremovable by pressure, which, added to the redness, gives the peculiar colour to the eruption. This is easily observed by pressing out the congestive redness, and when the latter disappears of its own accord a long persistent stain is left. There is some doubt how this pigmentation is brought about, whether by a breaking up of escaped red corpuscles or owing to some special tendency to the deposit of pigment, which is one of the peculiarities of the affection. I take, with Dr. Goodhart, the latter view, as I find that the rete is undoubtedly much pigmented although signs

of breaking down of red corpuscles are also apparent. It is to be noticed that the pigmentation in this affection is far more persistent than that which is noticed on the disappearance of an erythema multiforme, lichen planus, or a syphilide. As to the degree of rapidity with which the deposit takes place, more precise observations are necessary. In shape the eruption is primarily round or oval in outline, and the size is variously described as "uniformly that of a three-penny piece," a pea to a kidney bean, mostly a large pea to an almond, pea to a ten-cent piece, pea to a shilling, and end of cedar pencil. Larger and irregular eruptions or macules are formed by confluence. As the macules gradually get fainter the usually good definition of their borders is lost, and where they are very numerous, a mottling of the surface is produced. Thus in some cases there is a considerable variety of appearance met with in the lesions present. Generally the eruption is evolved with much itching, or continues irritable; but occasionally this is not a marked feature or is less noticeable after a time. As to the site the eruption occupies, no cutaneous region is exempt, but the trunk is early and prominently affected and next the limbs. It may evolve over the neck and face and even up the sides of the scalp in severe cases. The palms and soles are very rarely affected, and in the two most severe cases on record, the palatal and buccal mucous membrane was implicated. The affection seems to invariably pursue a chronic course by the evolution of the eruption singly or in crops, and the macules formed in this way may become so numerous in course of time as to occupy on the trunk and limbs as extensive an area as the intervening normal skin. The affection reaches its climax of intensity in infancy or early childhood, and after a variable period the evolution of eruption becomes less and less, and the urticarial or wheal-like lesions, if the latter term is preferred, gradually lose their two peculiarities of long persistence and pigmentation. The complete disappearance of all the stains has not yet been observed in any case, although the

near approach of this condition is evident in Dr. Tilbury Fox's three cases about ten years of age. In one of Pick's cases also the eruption had ceased to evolve at ten years of age, and Lewinski met with the eruption still well marked, but not very active, in a boy of eighteen years. The affection is not hereditary and is not dependent on any tuberculous, strumous, or syphilitic taint, nor is there any evidence forthcoming of any affection of the liver. The subjects of it are fairly healthy but perhaps not very robust, and the only point of importance is the excessive sensitiveness of the cutaneous vaso-motor apparatus as shown in the frequent occurrence of *factitious urticaria* and the lighting up of the older lesions by any emotional disturbance or derangement of the health.

The eruption was examined microscopically by Dr. Thin in Mr. Baker's case, and from the character of the cell-infiltration, its disposition, the gradual destruction of the gelatinous tissues, &c., he concluded that the morbid changes were indistinguishable anatomically from the earlier changes seen in lupus, whilst "from all other morbid processes which take place in the skin" it could be distinguished. That he thought it actually a phase of lupus appears evident from his remark that "it is impossible to resist the conclusion that had the child lived some of the affected parts would have ulcerated." We know now, however, that there is no tendency whatever to ulceration, but that the lesions subside spontaneously without leaving any scarring. Pick, of Prague, in a case which Lewinski hesitates to accept as genuine, removed a bright yellow, well-defined, oval wheal-formation, 8 × 3 mm., from the thorax. The sections disclosed little rust-brown hæmorrhagic centres embedded in the upper layers of the cutis, and surrounded by small cell-infiltrations. The papillæ were enlarged and the epidermis normal. It appeared to Pick that the pigmentation resulted partly from the long-continued hyperæmia and partly from hæmorrhagic exudation. In the central parts only the effects of the latter were noticeable, in the halo both.

Obstruction to the lymph-flow is suggested as another factor in the persistence of the lesions. When the boy Cornell was seven years old, I removed from his back, with the assistance of Dr. Bulkley, of New York, an old-standing, large, firm, raised, buff-coloured nodule, which I had made urticarial by rubbing, and hardened the specimens in chromic acid and alcohol. My sections display the formation of a typical wheal. A low power (Fig. 1) shows the fact that the morbid changes are limited to the upper third of the corium over circumscribed areas constituting the raised eruption seen on the surface of the skin. The changes reach their maximum in the centre of the area and subside somewhat suddenly peripherally, and in depth just above the level of the sebaceous glands. The changes are uniform over the involved area, and under a low power appear as a rarefication of the upper corium tissues with moderate cell increase scattered about uniformly. Under a high power (Fig. 2) it is seen that the epidermis is unaffected except that it is somewhat thinned from stretching, and that its lowest layer has a great excess of tawny pigment. *The tissue of the upper part of the corium is rarefied to the last degree.* Long strands of fibrous tissue radiate upwards from the deeper unaffected parts to be attached to processes of the rete, and from these fibres others branch and join to form a coarse network, the meshes of which are again divided and subdivided by delicate fibres and branching cells (see Fig. 2). These finer meshes contain a goodly number of cell-elements of different sizes and shapes, from mere nuclei to cells resembling white blood-corpuscles. Here and there capillaries, apparently of larger diameter than usual, are met with, but they are not prominent. Where the œdema subsides towards the periphery, larger blood-vessels come into view surrounded by rarefied tissues containing an increase of cells, and here and there little collections of golden pigment; and in these regions largely dilated lymphatics are seen. There is no thrombosis of the veins. The sweat-ducts and hair-follicles run down through this

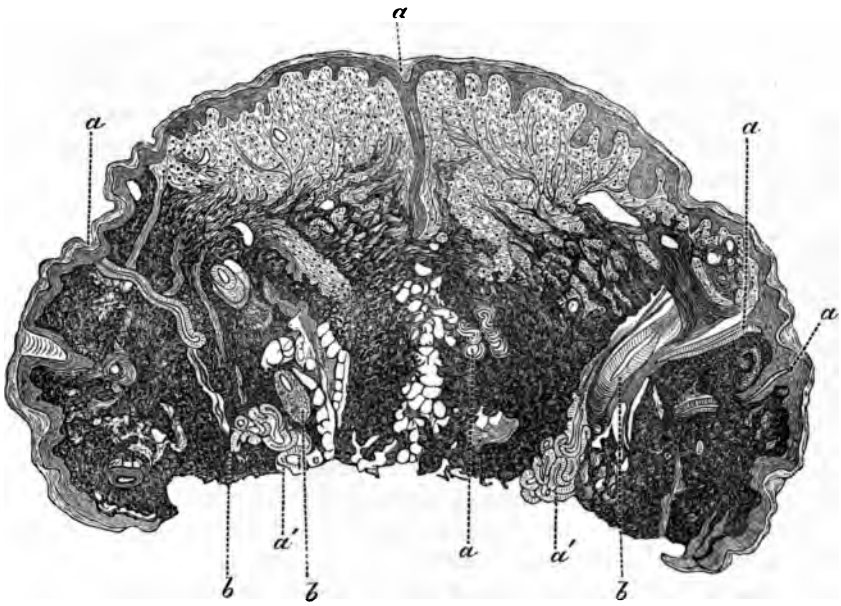

FIG. 1.—Vertical section. × × 75. *a*. Sweat ducts. *a'*. Sweat coils. *b*. Hair follicles.

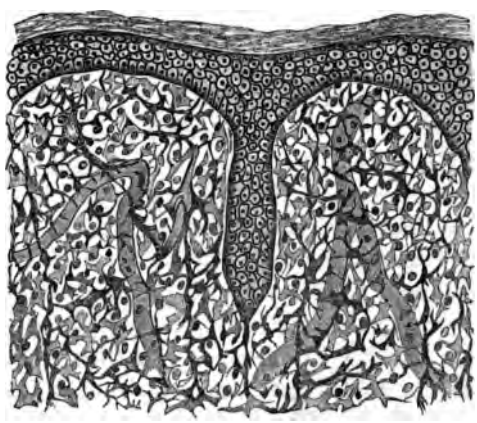

FIG. 2.—Vertical section of a portion of Fig. 1. × × 300. Showing the rarefaction of the œdematous corium.

tissue apparently unaltered to the equally unaffected sweat and sebaceous glands. I cannot make out any organisation of new tissue. The condition here met with is, indeed, only that brought about by prolonged œdema of the skin and corresponds closely to Renaut's description of urticaria in Cornil and Ranvier's pathological histology. Cell exudation was not very marked in my specimens, and certainly was not more than might be expected. Histologically, therefore, there is no doubt of the urticarial nature of the lesions.

The clinical difficulties in the way of at once recognising the eruption as a phase of urticaria are, (1) the rapid onset of pigmentation, which we have already discussed, and (2) the long persistence of the wheals. The existence of chronic urticaria in infancy and childhood is everywhere acknowledged, and in the affection formerly known as *lichen urticatus*, we have an urticaria presenting other special features. With regard to the pigmentation, I will only add the significant fact that Sangster in his case noticed the deposition of pigment in the factitious linear wheals excited by scratching in the great flexures. The persistence of wheals (urt. perstans) much beyond the ordinary time is not unknown though of rare occurrence. No allusion, however, is made to any special pigmentation (see for cases Willan and Bateman, A. T. Thomson, Tilbury Fox in 'Lancet' for March 8th, 1879, and Pick in the 'Prager Zeitschrift für Heilkunde,' 1881). Lastly, a most interesting case bearing on the subject was shown at the Congress in 1881 by Dr. Liveing in the person of a girl, aged about sixteen years, who suffered from mild urticaria for about a year and a half, and in whom the wheals, which were not noticed to be specially persistent, appeared to leave marked pigmentary macules which remained after the cessation of the urticaria.

I append in a tabular form a summary of all the recorded cases that I have been able to find.

Tabular Summary of Cases.

No.	Reference to case.	Age of onset.	Sites affected.	Description of eruption.	Complications.	Family history and general health.	Remarks of recorder, &c.
1	♀ *Nettleship, Brit. Med. Journ.*, Sept. 18, 1869, p. 323	3 months	At 2 years the pigmented spots thickly covered the neck and trunk, more sparsely the extremities, and slightly the forehead and margin of the scalp	Began as white "lumps" like the sting of a nettle, itching severely, and leaving brown stains on subsidence. At 2 years of age there were present: (1) non-raised brown stains, and (2) slightly raised from a little attendant congestion. Size uniformly that of threepenny-piece. No red wheals at this observation, although subsequently a number of erythematous and elevated patches were seen	A scratch with the finger produced in a few moments the ordinary urticarial wheal	—	—
2	♂ *Morrant Baker, Clin. Soc. Trans.*, vol. viii, 1875 (with plate)	6 weeks	First appeared on the back, and soon covered other regions; face affected last. At 12 months old very generally distributed; most profuse on back, rather less on scalp, face, and arms; none on palms and soles, and none near anus. Mucous membranes unaffected	Mother says the spots first appeared as small red pimples, which got larger. When first seen by Mr. Baker, Sept., 1874 (aged 12 months), there were papules and tubercles from a pea to a kidney bean, some nummular, some irregularly-oval; in some parts irregular wheals or patches from confluence of tubercles, 2–3 inches diameter, and raised about as much as severe urticaria, but smooth and flattened, feeling rather tougher than healthy skin. *Hue* dependent on varying degrees of congestion in different parts of the body, yellowish or yellowish-pink to decided pink or dull red. The yellowish spots do not fade on pressure, but the pink do. *Itching* only slight and occasional. The tubercles become urticarial on being rubbed. There was no material alteration until the death of the child when just over 3 years old. Fresh eruption had apparently ceased to evolve	—	Father and mother healthy. Child born at full term, well grown, always strong and well. One other child fairly healthy	Mr. B. says the difficulties are very great in grouping this affection with anything but *urticaria*, yet the persistence and inactivity of the eruption are unlike nettlerash. This child died of empyema, under Dr. E. B. Baxter's care, in March, 1877, at the Evelina Hospital, and then the eruption was in *statu quo*, neither advancing nor receding. Dr. Thin ('Clin. Soc. Trans.,' vol. x, 1877) examined microscopically the eruption, which was visible after death, and he concluded that the morbid changes were indistinguishable anatomically from the earlier changes seen in lupus.

No.	Reference to case.	Age of onset.	Sites affected.	Description of eruption.	Complications.	Family history and general health.	Remarks of recorder, &c.
3	Edward C. *Tilbury Fox, Clin. Soc. Trans.*, vol. viii, 1875; *and the present paper:* plate in *Atlas of Skin Diseases*	6 weeks	Appeared first on inner side of left thigh, then back of neck, and then the trunk. After 5½ months' duration the whole surface, including the scalp, penis, and hands and feet, were affected. The *mucous membrane* of palate and mouth was involved. In July, 1878, only the scalp, and palms and soles were exempt; the face was less affected than the trunk and limbs, where the eruption was exceedingly thick	Mother described the earliest eruptions as looking like "scalds," and she states that she could let water out from the "watery blisters." These original lesions persisted as stains for years. When first seen as an infant, in May, 1873, the surface was almost covered with buff-coloured nodules, interspersed with newly formed dull red blotches, and with pale fawn-coloured and slightly elevated patches, left in their turn by the subsidence of the nodules. In *size* they varied from split-pea to shilling or more, but very many were size of large pea, or size and shape of almond. In *colour*, a dull red or dusky copper at first, gradually getting buff; some occasionally pearly looking. In *consistence* feeling firm, like chamois leather. All considerably *elevated*. Surface uniform in some, but in most indistinctly marked, with a congeries of smaller projections, probably due to implication of follicles, giving a granular appearance. Recent lesions *itch*, often very much. In July, 1878, there were to be seen only dull brownish-yellow patches, in size from a split-pea to a five-shilling piece, the latter irregular from confluence. These maculæ are not raised now, and the red congestive hue is absent, except on the penis, where nodules are seen the size of the kernel of a hazel-nut. The appearance of the eruption is therefore very different to what it was five years previously. The lesions, however, acquire a distinctly urticarial character when briskly rubbed. The evolution of lesions has been comparatively infrequent for some time. "They come out as nettlerash." In Aug., 1881, the wheals still continued to appear now and then, leaving stains. In Jan., 1883, nothing but maculæ are to be seen, far less numerous and less deeply stained than formerly. The affection is evidently dying out. The boy often complains of itching and pricking, and when the mother looks she sees ordinary nettlerash. The wheals, however, are more evanescent, and do not appear to leave stains now	—	Father's family healthy; marked phthisis on mother's side. Child born at full time, and nothing particular the matter, but he has never thrived quite like the other children, who are all healthy. Jan., 1882: pallid, and not so well grown as the other children, but nothing specially wrong with him	In the earlier stages of this case (see *Atlas*) the eruption was much raised and nodular, and in some parts was indistinguishable in appearance from Xanthelasma, *e.g.* on the penis. The implication of the upper eyelids and palate increased the resemblance, but the patches here were not so closely like xanthelasma. Dr. F. came to the conclusion there was "clearly a deposit of buff-coloured stuff in the skin," and after insisting on the essentially capricious and temporary character of urticaria, he added, "I think, on the whole, that acute general xanthelasma would roughly describe the disease in my cases; but if it be thought that the disease is like, yet not the same as xanthelasma, I should be disposed to term it *xanthelasmoidea*." For microscopical examination, see present paper.
4	Dr. Brodie's	6 weeks	Trunk and limbs, and slightly on the face	When first seen as a baby in the summer of 1872, the eruption was altogether less marked and extensive	—	Good; no liver disorder; no con-	—

	case. ♂ *Tilbury Fox, Clin. Soc. Trans.*, 1875, vol. viii, and *Atlas*			than in Cornell's case, although the description of that case generally would apply to this. There was no pruritus. The eruption appeared as dull red patches, retaining colour and elevation for a long time, but eventually subsiding into tawny macules. At Christmas, 1874, the spots were disposed to lose elevation, and to assume a lighter colour. Jan. 4th 1883, Dr. Brodie frequently sees the boy, and the spots have almost entirely disappeared		stitutional symptoms; no syphilis in parents, who are both healthy	
5	Dr. Hebblethwaite's case, ♂ *Tilbury Fox, Clin. Soc. Trans.*, vol. viii, 1875; also *Atlas and present communication*	10 days	Trunk and limbs, most on chest, very few on the face	When first seen at 4 months old, in June, 1874, the eruption consisted of coppery, raised, granular patches, much as in Cornell's case. Jan., 1878, "better in every way." A fresh spot only now and then since first outbreak. There are numerous light mahogany-coloured patches and blotches over the body, from the size of a pea to a halfcrown, with an irregular but well-defined border, not raised now, and quite smooth. In March, 1883 (the boy just 9 years old), large, irregular, tawny-olive stains exist on limbs, trunk, and neck; especially marked on legs. They do not assume wheal aspect on rubbing. Fresh spots, with much irritation, have appeared from time to time when the child was out of sorts, but leave no stain now	"Still has nettle-rash occasionally" (note at 4 years old). The child constantly flushes from excitement or after food, and then the macules redden and get irritable. At 9 years old still subject to nettlerash	A blue-eyed, fair-haired boy, well grown and, tho' by no means robust, healthy. A peculiar and persistent hoarseness suggested hered. syphilis. Father died suddenly from unknown cause when a young man. Mother and her family are phthisically inclined; she had severe and frequent floodings before the baby's birth, Mar., 1883, distinctly nervous temperament; hoarseness has quite disappeared	In former notes of this case it was stated that some brown patches existed on the head and back at birth; but the mother is positive (T. C. F.) that the nurse drew her attention about ten days after the birth to irritable spots, which she now identifies with nettle-rash.
6	♂ *P. Albert Morrow, Archiv of Derm.*, vol. iii, 1876-7, p. 140, and vol. v, 1879	6 months	At 2 years old the spots covered the face and entire body, but most abundant on back and over flexures of joints; a few on palms and soles. The reddish spots appeared first on the back and chest, and soon the entire body was	At first a papular or tubercular eruption, looking distinctly elevated, and feeling so to the finger, from a pea to a ten-cent. piece in size; ordinarily pale-yellowish, changing to reddish or bright scarlet when the child was excited or cried; becoming urticarial when violently rubbed. In the 8–9 months under observation there was an obvious increase in the number of pigmented spots and of the irregularly distributed crops of tubercles or nodulated masses, which were most frequently observed on the upper part of the back. These nodules were	Has "hives" when the stomach is disordered, subsiding in 2–3 days. Urticaria attacks occur about once a week in warm weather, but far less frequently in winter; they	Well nourished, healthy-looking child; youngest of nine healthy children; never jaundiced	This case was exhibited to the New York Dermatological Society in Oct., 1876, for an opinion as to its nature, on account of its unusual features. It was called *Erythema tuberculatum*. I pointed out to Dr. Bulkley in 1878 that it was probably a case of urticaria pigmentosa, and Dr. Morrow

No.	Reference to case.	Age of onset.	Sites affected.	Description of eruption.	Complications.	Family history and general health.	Remarks of recorder, &c.
			covered with a persistent eruption. At 4 years old the entire surface of the head (except the superior portion of the scalp, the bridge of the nose and malar prominences), the trunk and limbs were covered. The mucous membrane of the palate and fauces were affected, but the hue of the eruption here was not yellowish	irregular in outline, as if from fused tiny nodules, elevated, in size from a coffee grain to an almond; when recent pinkish or bright red, and later becoming yellow or brownish; firm to touch. Some looked solid, as if containing lardaceous material. They developed suddenly, and *always* after an attack of urticaria; remained stationary for a time, and then rapidly subsided; average duration, 1–3 weeks, but sometimes longer. At 4 years old only 2 or 3 nodular masses were seen, but great increase in number of pigment spots, which coalesced on trunk into mottled eruption, losing their definite form. Colour from pink to yellowish-brown, deepening to livid red if skin is rendered hyperæmic; on the trunk a dash of green in them; on the forehead pale and indistinct. Mostly elevated, and overlying skin in loose wrinkles. Others less elevated seem studded with small flattened papules. The eruption irritable	are excited by irregularities of diet, emotions, &c. Similarly intense hyperæmia of the skin occurs. The skin is remarkably sensitive, and the slightest irritation produces wheals, *e. g.* exposure to air		afterwards published an elaborate account of the case with able comments. This severe case appears to bear a closer resemblance to that of Corneil than any other recorded. The nodular masses were a very striking feature in this case, especially in its earlier stages, and Morrow suggests their production by thrombosis of the lymphatic vessels preventing the escape of effused serum. He thinks each pigmented patch was probably the seat of a nodular swelling, and in answer to the question whether any once formed pigmented patches had ever disappeared, he says probably in course of time.
7	Caroline T *T. Barlow, Clin. Soc. Trans.*, vol. x, 1877	3 to 4 months	First on chest. At 13 months old, when first seen, front and back of chest and belly, front and back of arms, forearms, thighs, and legs, and the face. At 2 years old the hands and feet were still free	Uniform, brownish (with a dash of olive) pigmented patches, very slightly raised, in size from pea to shilling; unaltered during the year whilst under observation. One or two wheal-like eruptions occasionally seen. Never any ulceration	Said to be subject to flea-bites from early infancy, but Barlow never saw any true flea-bites. A scratch will bring out a *bonâ fide* wheal, and so will simple handling sometimes. Rubbing would always excite wheals from infancy	Phthisis in father's family, and two brothers of child died of acute tuberculosis. The patient never had jaundice, but she is tuberculous, and suffered from a crusted, ulcerating impetiginous eruption about the head and neck. No syphilis	"It must still remain a question whether recurrent vaso-motor disturbances of the skin are not a factor in their causation."
8	C. F. ♂ *Alfred Sangster*,	*Stains* first noticed at	The mottled red rash first appeared on abdomen, soon extending to almost cover	A coarsely mottled, red, irritable rash, gradually becoming browner in colour, *i. e.* buff-brown and buff-red. Some isolated, but for most part shaded off and blended with neighbouring macules. In	Ordinary urticarial wheals were seen from earliest infancy, but	Healthy-looking, well-nourished boy. Attacked with jaundice (?	"It may be conjectured that the disease .... is caused by vaso-motor changes in the skin, either occurring

	Clin. Soc. Trans., vol. xi, 1878 (with plate)	2 months old, but urticaria earlier	the trunk. At 2 years old the mottling had extended to the extremities; thickeston back, abdomen, and sides; the surface in later regions almost uniformly brown; also backs of arms and forearms; loins and buttocks least affected; face (except right cheek slightly), scalp, backs of hands, palms and soles free; big flexures (where scratching is most vigorous) specially affected	the big flexures brown patches are in lines, parallel with the natural folds of the skin, and following scratching apparently. Some patches faintly raised, but no infiltration of the natural textures. The skin only a little coarser over the patches. The buff-red patches lose red element on pressure. At 2 years old the older patches on abdomen had lost red colour and become browner	left no stains at first, according to the mother's statement; they were excited by any irritation, especially scratching, and this tendency was marked throughout; simple handling brought out wheals. Sangster saw linear and other wheals resulting from scratching whilst examining the child	spurious) before a week old, which lasted a fortnight. Another child, who died at the age of six weeks, said to have been jaundiced. No abnormal stools; no bile or sugar in urine; no syphilis in father or mother. Psoriasis in family	spontaneously, or set up by external causes. . . . The vaso-motor changes are possibly connected with functional liver disturbance; at any rate they have a tendency to become permanent and lead to further pigmentary changes."
9	Frederick P. *T. Barlow, Clin. Soc. Trans.*, vol. xii, 1879	9 weeks	About a dozen spots first on chest, belly, back, and upper arms; gradually he became covered over these regions. At 8 months old, trunk, especially the back, the thighs, and arms, neck, and the temples where the hair began, and a little way into hair	Some of first crop of spots "were like large blisters, red all round," and they were irritable. At 8 months old, when first seen, brownish-drab, rounded areas, the size of end of cedar pencil, many confluent; finely granular surface and slightly raised, more so on the trunk	The boy was ready to scratch his trunk directly it was exposed. Had "white blister-like spots" (? wheals) come out from time to time. "It is always easy to produce a band of hyperæmia by a scratch, and this band often shows a wheal-like line along its centre." Occasionally an ordinary wheal was to be seen	Bright, active child, with no sign of visceral disease. Father and mother healthy. No syphilis. Child never jaundiced. Some constipation. Another child healthy	—
10	Esther B. *Goodhart, Med. Times and Gas.*, Feb. 1, 1879	5 months	When first seen, aged 7 months, greater part of trunk involved, chiefly stomach and back; arms, less on lower extremities; back of head, ears, and round neck; none on face, hands, or feet. First appeared on abdomen	At first small, irritable, whitish elevations, surrounded by a reddish blush. The lesions continued to appear as urticarial wheals for the 4 months whilst under observation. The white elevations have a reddish halo; they quickly acquire a livid brownish staining, and subside into persistent, deep brown, rather sharply defined, slightly raised, rugose, non-indurated, round, oval, or irregular patches, or semi-organised wheals	Not due to flea-bites	"Perfectly healthy looking child." No syphilis; no jaundice	The pigmentation "ensues so rapidly after the appearance of the wheal, as to make it probable there is some peculiar tendency in this child to the deposition of pigment on slight irritation of the skin." Dr. Goodhart has seen other cases, to which he briefly refers.

No.	Reference to case.	Age of onset.	Sites affected.	Description of eruption.	Complications.	Family history and general health.	Remarks of recorder, &c.
11	Solomon G. *Pye-Smith, Guy'sHosp Reports*, 3rd ser., vol. xxv, 1880-1	3 months	Under observation, at 2 years old, greater part of back, chest, abdomen, and adjacent parts of arms and thighs, hands and feet covered with the papules; head and neck free	Rather large, discrete, yellowish-brown papules. Two or three fresh wheals seen, with erythematous injection around	—	—	Sent by Dr. Goodhart to Dr. Pye Smith. Rash not affected by teething.
12	♂ Alfred P. *Cavafy, Lancet*, May 8, 1880, vol. i, p. 739	14 days	Commenced on back, and thence quickly spread over trunk. At 15 months old, when he came under observation, lesions largest and most numerous over the back and chest; fewer but larger on abdomen; many over buttocks, upper and lower extremities (except palms and soles); smallest on neck, sides of face, and temples; 2 or 3 large ones over occipital and parietal bones	Said to have commenced as flat red patches on which blisters formed. Urticaria bullosa and crusting seen occasionally. At 15 months old, large, rounded or oval, flattened patches varying in colour from bright or dull red, with a tinge of yellow, to orange or pale buff. The red colour depends on different degrees of hyperæmia, which varies according to situation and age of the lesions Pressure removes the hyperæmia and leaves a buff stain. No itching except after mechanical irritation. Shape rounded or irregularly oval, some 3–4 inches long by confluence. On the neck they are more or less parallel to the transverse folds of skin, and slightly raised. Most lesions are barely if at all raised; their texture is a trifle coarser than normal skin, but there is no infiltration. An urticarial condition is revived in the lesions by irritation but not easily in the stains	Spurious jaundice soon after birth for one month (urine and fæces normal). Wheals often present, especially around neck and waist, and seen by recorder. Linear wheals can always be excited by scratching. Crying and struggling increases hyperæmia of lesions	Born at full time; fair haired; general health excellent throughout, well nourished and robust at 15 months. No syphilis and no phthisis in family. Another child healthy. Parents healthy	Shown at Harveian Society, April 1, 1880. Under observation 3 months.
13	A. F. ♂ *Stephen Mackenzie, Med. T. and Gaz.*, April 24, 1880	3 days	At first over chest, stomach and back, and subsequently spreading slightly to neck, inner side of thighs, and to forearms, when first under observation at 15 months old	At first non-irritating, large, dark brown, non-elevated patches *in* the skin, many larger by confluence. Since evolution persisted unaltered or a little paled. At 15 months old buff, or buff and pink, slightly raised islets, from a pea to irregular patches size of sixpence. They become raised and redder by rubbing	Not specially bitten by fleas, &c. Any irritation, such as scratching or washing, excites urticaria	Healthy-looking child, born at full term; never jaundiced. No syphilis in family Rest of children healthy	Dr. M. supposes by *dark brown* the mother probably meant *red*, as is often the case amongst the poor. He considers the affection undoubtedly *urticarial* in nature, and the buff colour to be produced by diapedesis of coloured blood-corpuscles during the wheal stage, and subsequent disintegration and staining of the skin.

							There must be a certain amount of hyperplasia of tissues to account for the persistent swelling.
14	Sex ? *Stephen Mackenzie, Hunterian Society's Reports,* 1880-1	3 days	When first seen, at 16 months old, eruption limited to the trunk, back, and front, the neck and upper part of thighs	First appeared as red spots, and persisted ever since. No irritability in the skin till the last month (15 months old). At 16 months buff-coloured raised patches, becoming urticarial when scratched, and subsiding into buff patches in about 15 minutes	A small bulla seen in one place	—	—
15	G. F. ♀ aged 8 years. *F. J. Pick, Prager Zeitschrift für Heilkunde,* 1881	6 weeks	—	It was stated that the affection commenced with a well-marked, raised, wheal-like eruption, which persisted a long time and left a discoloration, and was ushered in by fever. Similar outbreaks were repeated at longer or shorter intervals, but never so long but that the discolorations left by former wheals remained. No febrile disturbance accompanied the later outbreaks. Itching was violent in first attacks, but absent later on, so that fresh eruptions were only noticed by the aspect of the skin. This state of things continued until August, 1879, when the child, then aged 8 years, was first seen by Pick. In September, 1881, the evolution of eruption had ceased, but rounded, elevated, and flattened brown-red spots, 3–5 cm. diam., remained, either uniformly coloured, or with the central part brownish red and the periphery shaded off into other brownish or yellowish tints. Pick observed that the evolution of the eruption was rapid, and followed sometimes on a slight pressure of the skin, but resolution was long delayed, for they were still raised after 2–3 weeks, leaving in 6–8 weeks a brown-yellow spot. Never any scaling or pus formation	—	Father well made and healthy; mother died after birth of a 6th child of puerperal septicæmia; other children fairly healthy. No tuberculosis. Patient (2nd child) well developed, fair haired, blue iris, and little general pigmentation	—
16	K. G. ♂ *F. J. Pick, Prager Zeitschrift für Heilkunde,* 1881	A few days after birth	Back, breast, abdomen were thickly sown, extending later, but to a less degree, on upper extremities, and sparsely to lower limbs; slight on the face	Pick repeatedly traced the formation of the eruption thus:—A hyperæmic spot, 3 to 5 cm. long, appeared suddenly (often upon uncovering the surface) and with great itching, which, however, soon subsided. Its centre became *rapidly* the seat of an oval wheal, ¼ to 1½ cm. long by 3–5 cm. wide, and raised 1–2 mm. This central part was darker coloured than its halo, and firmer. By the continued evolution of such eruption the surface of the skin became covered. When first seen in Aug., 1880, at 7 months old, the skin of the body,	—	Well-developed fair-haired boy, with grey irides; anæmic; general health fair till contracted variola at 15 months old	"No cause lying outside the organism can lay claim to its origin." Pick removed a bright yellow, well-defined, oval wheal formation, 8 mm. × 3 mm., from the right side of thorax. Sections disclosed little rust-brown hæmorrhagic centres embedded in the upper layers of the cutis, and surrounded

No.	Reference to case.	Age of onset.	Sites affected.	Description of eruption.	Complications.	Family history and general health.	Remarks of recorder, &c.
				especially the back, was covered with more or less pigmented spots of two kinds: (*a*) hyperæmic red macules (not raised), from which a certain amount of hyperæmia disappeared on pressure at the periphery, and left a brown discoloration in the centre; (*b*) a preponderance of raised wheal-like formations, not painful, firmer than natural, ½ to 1 cm. diam., of a sulphur-yellow to a bright orange colour; colour, bearing the closest resemblance to xanthelasma, not disappearing by pressure. Between the colours here mentioned all shades were seen significant of blood extravasation in the skin. No signs of scratching; no desquamation; no pus formation			by small cell-infiltrations. The papillæ were enlarged, the epidermis normal. It appeared that the pigmentation resulted from long-continued hyperæmia on the one hand and from hæmorrhagic exudations on the other. In the central part the hæmorrhages were seen apart from the effects of long-continued hyperæmia, in the halo both together. Pick surmises that obstruction to lymph-flow is another factor in the persistence of the wheal. Lewinski expresses a doubt whether this was a genuine instance of what has been described as urt. pigmentosa.
17	♂ *Lewinski, Virchow's Archiv*, Bd. 88, 1882	5 weeks	First appeared on abdomen, and at the age of 9 years had gradually extended over the whole body, including the soles of the feet. At 18 years old, however, palms and soles free	The mother relates that first a nodule, the size of a three-penny-bit, was seen, followed by other similar ones near, and also having vesicated summits, and of very short duration (a few days), leaving pigment spots of a very persistent nature. The eruptions felt firm to the touch and quickly disappeared, but there was a recrudescence of some spots. At 9 years old nodules appeared similar apparently to the others, but not leaving stains. The stains gradually became paler on the face and upper arm, but elsewhere did not alter. The patient came first under observation *at the age of* 18 *years*, and then similar eruptions were appearing. At this age his skin was covered with lentil-sized, or rather larger, rounded or oval spots, disseminated, unaltered by pressure, and pale yellow, on face, neck, and extremities, but numerous, a little raised, and confluent on trunk, especially chest, where they form a coloured net-work, enclosing small islets of normal skin. On the back	Violent itching from earliest childhood, and a special sensibility to cold, so that he always sat in by the fire. In his youth he suffered from cold hands and nose, and chilblains. With any medium access of warmth, however, he became scarlet and swollen in the face. Urticarial wheals were easily pro-	Healthy parents and five healthy brothers and sisters. Patient always strong and well. Urine normal	Under observation for a few months until the patient left Berlin. Treated internally with atropine and externally with a 1 per-cent. solution of carbolic acid with some amelioration.

	Clin. Soc. Trans., vol. xi, 1878 (with plate)	2 months old, but urticaria earlier	the trunk. At 2 years old the mottling had extended to the extremities; thickeston back, abdomen, and sides; the surface in later regions almost uniformly brown; also backs of arms and forearms; loins and buttocks least affected; face (except right cheek slightly), scalp, backs of hands, palms and soles free; big flexures (where scratching is most vigorous) specially affected	the big flexures brown patches are in lines, parallel with the natural folds of the skin, and following scratching apparently. Some patches faintly raised, but no infiltration of the natural textures. The skin only a little coarser over the patches. The buff-red patches lose red element on pressure. At 2 years old the older patches on abdomen had lost red colour and become browner	left no stains at first, according to the mother's statement; they were excited by any irritation, especially scratching, and this tendency was marked throughout; simple handling brought out wheals. Sangster saw linear and other wheals resulting from scratching whilst examining the child	spurious) before a week old, which lasted a fortnight. Another child, who died at the age of six weeks, said to have been jaundiced. No abnormal stools; no bile or sugar in urine; no syphilis in father or mother. Psoriasis in family	spontaneously, or set up by external causes. . . . The vaso-motor changes are possibly connected with functional liver disturbance; at any rate they have a tendency to become permanent and lead to further pigmentary changes."
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10	Esther B. *Goodhart, Med. Times and Gas.*, Feb. 1, 1879	5 months	When first seen, aged 7 months, greater part of trunk involved, chiefly stomach and back; arms, less on lower extremities; back of head, ears, and round neck; none on face, hands, or feet. First appeared on abdomen	At first small, irritable, whitish elevations, surrounded by a reddish blush. The lesions continued to appear as urticarial wheals for the 4 months whilst under observation. The white elevations have a reddish halo; they quickly acquire a livid brownish staining, and subside into persistent, deep brown, rather sharply defined, slightly raised, rugose, non-indurated, round, oval, or irregular patches, or semi-organised wheals	Not due to flea-bites	"Perfectly healthy looking child." No syphilis; no jaundice	The pigmentation "ensues so rapidly after the appearance of the wheal, as to make it probable there is some peculiar tendency in this child to the deposition of pigment on slight irritation of the skin." Dr. Goodhart has seen other cases, to which he briefly refers.

TWELVE CASES

OF

TUMOUR OF THE BLADDER

(10 IN THE MALE AND 2 IN THE FEMALE),

IN ALL OF WHICH AN

OPERATION HAS BEEN PERFORMED FOR THE REMOVAL OF THE GROWTH,

WITH THEIR HISTORIES AND THE RESULTS.

BY

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ETC., ETC.

Received May 5th—Read June 12th, 1883.

I DESIRE to present to the Society a brief account of seven cases of tumour of the bladder which I have operated on since the reading of my previous paper in January last (1883), when the histories of four other cases were read. Further, it may be remembered that in the preceding year I showed a patient from whom I removed a vesical tumour now more than two years and a half ago. A group of twelve cases is thus formed, and they furnish data which may be useful in guiding us, to some extent, in observing and treating similar cases in future.

A brief *resumé* of the preceding five cases may be given as follows:

Case 1.—T. R—, æt. 29. A single pedunculated papillomatous growth, removed by perineal incision in November, 1880. He made a rapid recovery, and is now in perfect health.

Case 2.—Mrs. F—, æt. 30. A rather large papillomatous tumour removed in May, 1882. She is now in perfect health. A carefully-made histological report by Mr. Stanley Boyd is annexed.

Case 3.—B. G—, æt. 46. Had a large tumour filling the bladder. I removed the greater portion, and he died a day or two after. The bleeding was very severe. The growth was examined by Mr. Boyd, and his report thereon is now appended.

Case 4.—Dr. M—, æt. 52. I removed a considerable papilloma in November, 1882. The patient was present at the meeting of our Society when the last paper was read; his own being one of the cases. He has recently had slight return of the bleeding after a long walk of several miles, but is otherwise well. A report by Dr. Gibbes is appended.

Case 5.—K. G—, æt. 67. In January, 1883, I removed a firm sessile growth from near the base of the bladder; and it is now at the museum of University College. It has been examined by Mr. Shattock, who has favoured me with the report which is appended.

The following cases, now reported for the first time, form the second series:

Case 6.—T. F—, æt. 67. January 17th, 1883. About three years ago he first saw blood in the urine; no return of blood until four months ago, since which it has been constant and gradually increasing. He has several times observed the stream to begin clear, and become red as it passed; and it is mostly more bloody at the close. This I have myself witnessed. He passes urine three or four times in the night, holds it three or four hours in the

day; has some smarting in the penis, and has pains in the back when taking exercise and at other times. Shreds passed in the urine contain abundance of long spindle-shaped cells, running into fibres.

Operation on January 30th.—After the usual incision a growth was at once recognised; large, broad, and occupying the left side of patient's bladder, near to the neck, extending far back. With time and patience I extracted a large quantity of rounded masses of growth, rather firm in texture, and tied in a tube as usual. Pain and bleeding not considerable.

February 3rd.—Very slight bleeding; urine improved; patient doing well.

5th.—Bleeding completely ceased.

6th.—Removed the tube.

15th.—Retains urine three hours; no pain.

21st.—Wound healed; went home, feeling very well.

March 13th.—Slight frequency of micturition, very slight pain; a trace of blood seen now and then in urine.

April 9th.—A short attack of bleeding four days ago. To inject solution of perchloride of iron.

The tumour was examined by Dr. Heneage Gibbes, whose report is appended. It is epithelioma, associated with some villous growth.

CASE 7.—W—, W. æt. 63. January 24th, 1883. Seven years ago he first suffered from bleeding, and retention caused by clots; a condition which has been repeated at long intervals ever since.

Last year numerous bleedings. Last month much blood lost; bleeding less when no exercise; not much pain. Rises two or three times at night; retains urine two hours in the day. Often sees urine clear at commencing and very red at close of the stream. He passed an excellent specimen of villous tumour at the first visit.

February 8th, 1883.—I operated, readily finding a tumour, rather sessile, with a wide base, at back of bladder towards right and on the floor adjacent. All the

salient portions removed, leaving rough or slightly ragged base. A tube tied in.

13th.—Feverish; took out tube.

20th.—All urine passes by wound, still bloody.

March 2nd.—Sitting up and moving about. Very slight blood, this lessened by injection of perchloride of iron into the bladder.

12th.—No blood seen for a week; holds urine three hours. Walked out for an hour yesterday.

April 3.—Has been three weeks at home; a slight frequency of micturition, pain trifling, no blood, and feels better than he has done for a long time; has gained flesh and strength.

June 6th.—Living and well.

The tumour was examined and regarded as villous papilloma, and by Dr. Heneage Gibbes whose report is appended.

Case 8.—J. M—, æt. 64. February 10th, 1883. First saw blood in the urine about a year ago; last six months this symptom has much increased, and pain and frequency have appeared also; the last three months the blood has been continuous and excessive in quantity, so that he has been confined to his room during that period. For several days examined the urine-deposit microscopically, and found no villi, but only large, oval, granular cells. But his condition called for further research, and I determined to explore the bladder.

February 21st.—Subject very stout, and perineum deep; on entering the bladder my finger encountered on patient's left a wide-based sessile growth, very hard and firm, mammillated, with two heads projecting above the rest. I removed the greater portion with some difficulty, leaving a thickened base, and tied in the tube as usual.

24th.—Took out tube; blood still free.

March 3rd.—Urine slightly bloody; about half passes by wound. No pain; takes food well.

6th.—Blood ceased to day.

16th.—The wound has healed; urine free from blood; takes food well; holds water two hours.

April 18th.—No blood since; slight cystitis; complains chiefly of pain and swelling under left thigh. There is a tumour there, suggesting suspicion of secondary growth. Health fair.

May 5th.—The patient has been suffering severely with pain and increasing swelling of the tumour of the thigh, and now difficulty in micturition has appeared, due to the impaction of a phosphatic concretion in the urethra, and this was speedily relieved. It has been occasioned doubtless by an increased irritability of the bladder which has arisen of late; still no blood has been seen in the urine.

Case 9.—Mrs. ——, æt. 65. February 22nd, 1883. She first noticed blood in the urine about seven or eight years ago; has had much pain in the left side and back. Many severe attacks at different times since, with intermissions of bleeding for two months.

November, 1882.—A severe attack, which has never ceased since, in spite of much styptic treatment; very slight pain in left side and none in the bladder; micturition frequent, not painful, the urine is offensive, bloody, mucopurulent; sounded but nothing felt. Examination of the urine shows abundance of spindle-shaped cells and fibres.

February 23rd, 1883.—Dilated the urethra and found tumour occupying base and right side; much time spent in carefully nipping off a large quantity in small masses; bleeding very free.

28th.—Much pain in the night, very little bleeding; no urine passed, only three ounces removed by catheter. Pulse 100, temp. 99°. Some vomiting.

March 1st.—No urine has been passed; abdomen tender; takes some food. She died in the night with complete suppression of urine.

2nd.—*Autopsy*.—No trace of peritonitis, both kidneys

much diseased; the left contains a considerable calculus, with marks of long-standing inflammation and suppuration around it.

Mr. Eve of the College of Surgeons examined the growth and reported it to be "a villous papilloma."

Case 10.—J. S—, æt. 53. First seen on August 24th, 1881. For six months past has been subject to frequent micturition with pain before and after the act, not worse for movement, occasionally a slight bleeding seen. Urine examined by microscope, nothing but blood, no *débris* of growth.

May 8th, 1882.—Came up again. Sounded the bladder and found nothing but a soft velvety surface, as if coats thicker than natural (the only physical sign sometimes presented by tumour); prostate rather full from rectum. Pain and frequency the same, worse at night; not aggravated by activity. Repeated careful examination by microscope discovers no tissue *débris*.

August.—Symptoms nearly the same, rather more pain, but florid blood seen at the end of micturition.

November. — Symptoms much aggravated, nothing characteristic found in urine. Heard in December that he is decidedly worse.

March 2nd, 1883.—Last two months has bled every day, and passes urine every hour with much pain in the penis, none in the legs or back. Appetite fair; walks one or two miles daily; has lately become thinner.

3rd.—Operation found large mass half filling the bladder and harder than any I had felt before. It sprang from the opposite base and a little from the patient's right side. I removed the growth with a cutting forceps only, owing to the density of the structure, bit by bit; bleeding very free; a tube was put in; pain was inconsiderable.

4th.—Supra-pubic pain and sickness; urine passes freely by tube and is bloody.

5th.—Less blood; very weak, pulse 130. Abdominal pain; takes milk, champagne, &c.

6th.—Very slight difference to report.

7th.—Much weaker, restless. Died at noon.

Autopsy.—A portion of tumour on right side, very firm, was still remaining; the adjacent parts of the bladder seem to be infiltrated with tissues like the growth itself. Both kidneys small and pale, the right showing signs of pyelitis. Lumbar glands are slightly enlarged. It is worthy of remark that no *débris* was ever found indicating the presence of this growth, although repeated examinations of the urine deposits were made.

Mr. F. S. Eve examined this specimen and reported as follows:—"The tumour consists of alveoli, enclosed by their connective tissue bands, and containing elongated and columnar cells, and being therefore an example of a cancerous growth."

Case 11.—W. D—, æt. 65. February 8th, 1883. Nearly one year ago the patient first saw blood in the urine; it has been more or less constant ever since. Urine of a brownish tint, with shreds; these under the microscope show abundant fusiform cells and fibres.

March 30th.—Operation: on introducing my finger I encountered behind the neck of the bladder, on the floor and spreading to the patient's right, several eminences formed by a growth, firm, sessile, without pedicles. Some of these I snipped off with cutting forceps, leaving the chief part, a hard mass, occupying the base, and apparently involving the walls of the bladder, as far as I could estimate with one finger in the bladder and the other in the rectum. Put in the tube as usual.

April 2nd.—Bleeding ceased, took out the tube.

5th.—Patient is feverish and weak. He continued thus, never losing blood, but gradually becoming weaker, and dying on the 12th inst., thirteen days after the operation.

This is the first tumour which, on account of its form, I desisted from endeavouring to remove. It was examined by Dr. H. Gibbes, who reported as follows:

"The tumour arises from a solid base and is only in a small part villous growth. The base contains non-striped muscle, and is probably a direct outgrowth from the walls of the bladder, which it exactly resembles. Proceeding towards the surface in the submucous tissue, a number of inflammatory foci occur as round masses of small cells, while directly under the epithelium the whole of the submucous tissue is infiltrated with inflammatory cells and a number of capillaries run directly towards the surface. The epithelium on the surface is similar to that of the normal bladder, this description applies to the main body of the tumour. There are, however, several small outgrowths from this, which resemble the villous growths examined before; they have the connective-tissue trabeculæ, with stratified columnar epithelium arranged on them, in some bases there are crypts lined with epithelium. With regard to the question of malignancy, this tumour has only the elements of the normal bladder, which are arranged much in the same manner as usual. There are, however, some small patches of large cells that look very suspicious, but there is nothing to enable me to speak positively as to its malignant character."

Case 12.—C. C. S—, æt. 56. January 11th, 1883. About one year and a half ago he had for the first time severe pain in the neck of the bladder and urethra after passing water. Three months ago first saw blood in the urine after a walk.

Present state.—Great irritability and pain, and frequently observes blood at the end of passing water. Sounded: nothing detected, the bladder empties itself by its own efforts; nothing felt by rectal examination. The urine shows no sign of tumour *débris*. He returned to the country to carry on certain plans of treatment there. I heard that he occasionally passed phosphates, and that the symptoms increased in severity.

April 3rd.—Came up again. Sounded under ether: no enlargement of the prostate, the "soft velvety" feeling

of the bladder was notable. To try injections of iron for the bladder, &c.

During the month of April much bleeding and pain; small concretions of phosphate passed. I decided to open the urethra for the purpose of resting the bladder and allowing the urine to drain off for a time.

May 4th.—On introducing the finger felt a large sessile mass, formed by a non-pedunculated tumour occupying (patient's) right side and base of bladder, but evidently one which there was no possibility of removing; incorporation of tumour with walls of the bladder appearing to be complete. I nipped off two more salient portions than the rest, and sent them to Dr. H. Gibbes for examination.

5th.—Urine passing well and almost without blood stain through the tube. Patient more comfortable than before the operation.

Dr. Gibbes' report on this case is appended; he regards it as resembling a "hypertrophy of the submucous coat of the bladder; no villous growth is present."

Remarks.—The operation has in all cases been performed as described elsewhere,[1] and consists in opening the membranous urethra from a small median incision in the perineum, and in dilating the prostate gently and gradually with the left index finger, which when fully introduced explores the entire internal surface of the bladder, while supra-pubic pressure is made under complete anæsthesia. The way is thus made for the introduction of the forceps. Of these I have designed and employed two or three forms, and found them useful to meet varied forms of growth; and some of these are now provided with cutting edges. I may remark that while in some cases the prostate is dilatable, and admits the finger without receiving much if any injury, in others a certain degree of rupture is inevitable. This I have recognised during life, and have witnessed in two of the

[1] 'Proceedings,' New Series, vol. i, p. 51.

fatal cases; but the division of tissue thus produced is not considerable.

In estimating the results of the proceeding—which it may be stated has, in every case but the first, consisted of simple external urethrotomy of the membranous portion—it is necessary to recall the fact that unless removed by surgical operation, vesical tumour is *inevitably fatal. Every recovery is a clear gain; and a fatal issue is simply the natural termination forestalled.* In these twelve cases there have been five recoveries; and in some of these there is every reason to believe that the cure is permanent, since there is no sign of recurring symptoms. In three cases death succeeded the operation in a few days. In one it occurred in a few weeks. In the remainder, the pre-existing severe and continuous hæmorrhage has been completely checked for a period of some months, and occasional slight bleedings have reappeared. The future course of these cases will be closely watched and duly reported.

Such a result I would submit is one which warrants a cautious observation of the numerous cases of hæmaturia which are to be found, and also the employment of digital exploration of the bladder, when the facts ascertained indicate that the cause of bleeding is not due to ordinary, obvious, and well-known conditions.

When an adult, either male or female, has long been the subject of unduly frequent micturition, the act mostly painful, with occasional hæmaturia, this sign increasing gradually in importance as the case advances, and furnishing evidence meantime that its source is vesical and not renal, a physical, *i.e.* surgical examination of the organs is of course indicated. The result of this may show that no calculus is present, and that no obvious enlargement of the prostate or of the adjacent structures can be affirmed. Nothing in the history can be distinctly referred to as a cause; no clue in fact is offered, except such as may be found by repeated examination of the urine. And it is by this particular means that the most

trustworthy information is to be obtained. But before describing the evidence furnished by the urine, I will refer to the facts which may be obtained by the study of these twelve cases. I have therefore arranged the chief points worthy to be noted in a table, so that they may be seen at a glance.

The facts referred to are as follows:

1. *Nature of the growths.*—Eight were examples of simple papilloma; more or less associated with dendritic prolongations of the villi natural to the vesical mucous membrane. When the growth consists entirely of these elongated villi, the term "villous tumour" suffices to describe them. Three consisted of growths, more or less malignant; *e.g.* epithelioma and formation of cell-growths allied to sarcoma. These also are associated with villous development like that noted in the preceding class; and in two instances, there was deposit in the neighbouring glands. One was a product closely resembling the submucous tissues of the bladder without villous growth.

2. *In relation to duration of symptoms.*—It is evident that the course of papilloma is slow. The symptoms are recorded as occupying periods before operation, varying from three to seven years. I have known longer terms still in cases formerly observed, and left in the natural course of events to end in death, as they invariably do, if not relieved by surgical aid. But the progress of malignant growth is more rapid in the bladder, as it is elsewhere; epithelioma as usual being the slowest of production in the class; from one year to two years and a quarter are the terms presented by the examples in our table.

3. *In relation to diagnosis.*—A notable fact in the history of papilloma and of villous growths is, that the appearance of blood in the urine is one of the earliest, if not the earliest, signs of derangement in the urinary function observed by the patient. On the other hand, in the malignant growths met with, painful and frequent micturition have long preceded the appearance of blood

Table of Twelve Cases of Operation for Vesical Tumour, by Sir Henry Thompson.

No.	Case.	Age.	Duration of symptoms.	Earliest sign observed.	Result of urine examination.	Complication with calculus.	Nature of tumour.	Result of operation.
1	T. R.	29	3½ years	Blood in the urine	Not examined, no growth being expected, the operation was made for a supposed encysted calculus	A small oxalate of lime and phosph. calculus crushed	Papilloma	Living and well.
2	Mrs. F.	30	6 „	„	Ditto	—	Papilloma villous (Mr. S. Boyd)	Living and well.
3	B. G.	46	1 year	Frequent micturition; blood much later	Much large cell-growth, various forms	—	Fibro-sarcoma (?) (Mr. S. Boyd)	Died few days after operation: no autopsy: probably some giving way of bladder at base of tumour.
4	Dr. M.	52	5 years	Blood in the urine	Shreds formed of fusiform cells	—	Villous papilloma (Dr. H. Gibbes)	Well nearly six months, recently a second operation, since which no bleeding.
5	E.K.G.	67	6 „	„	Ditto	A small uric acid calculus crushed	Papilloma; resembling structure of "soft warts" (Mr. Shattock)	Returned to the Cape: probable reappearance of the tumour.
6	T. F.	67	3 „	„	Numerous fusiform cells and fibres	—	Villous epithelioma (Dr. H. Gibbes)	Recent signs of reappearance of tumour.

7	W. W.	63	7 „	„	Well-marked villous growth	—	Villous papilloma (do.)	Living and well.
8	J. M.	64	1 year	„	Never found any characteristic débris in urine	—	Composed of normal bladder tissue with villous structure added; no structure resembling malignant growth was found	Died two months after with secondary malignant growth in thigh.
9	Mrs. O'R.	65	7 years	„	Large spindle-shaped cells	Calculus in left kidney, large; both kidneys diseased; pyelitis	Villous papilloma (Mr. Eve)	Died three days after with suppression of urine.
10	J. S.	53	2¼ „	Frequent and painful micturition. Blood at later stage	No characteristic débris found	—	Villous cancer (do.)	Died few days after operation.
11	W. D.	65	1 year	Blood later	Numerous long cells and fibres	—	Tissues like those of the walls of the bladder, and some villous growth in small quantity on surface (Dr. H. Gibbes)	Died fourteen days after operation, of exhaustion.
12	C. C. S.	56	2 years	Pain first; blood later	Nothing found	—	Tumour not removable; a portion taken away is regarded as resembling a "hypertrophy of the sub-mucous coat of the bladder: no villous growth is present" (Dr. H. Gibbes)	Living: symptoms relieved at present; probably from drainage of bladder.

Total result, seven recoveries, more or less complete, and five deaths.

in the urine. The same is of course true in cases of calculus and of prostatic disease.

In all vesical tumours the patient observes, as the disease progresses, that the stream of urine contains a larger admixture of blood at the close of the act than at the beginning. It often happens that the stream may commence with pure unstained urine, and become florid towards the close; and this bright red is the usual tint, and not the brown shade common in other conditions.

The microscopic examination of urine is of great importance in relation to diagnosis. In cases of villus and papilloma, and indeed of all tumours, the microscope is of great value. After my recent experiences, I attach much greater importance to it than I formerly did. At the same time it is not difficult to be deceived by some of the products found in the urine of patients where no serious organic changes have occurred.

First let me say that the deposit from a patient's urine should be examined in a fresh state; search being especially made for shreds which are mostly passed at the end of the stream, or which may be sometimes washed out by a plain water injection through the catheter. It may be necessary to repeat this examination several times. In Case 7, I found in the urine passed at the patient's first visit to me, one of the most perfect specimens of villous growth I ever saw. A slender, club-shaped process with a complete covering of columnar epithelium, and in the centre of the stem its blood-vessels full of red corpuscles clearly seen. After this, on four consecutive days, as many examinations were repeated but absolutely without result. The first observation, however, sufficed to assure me of the presence of tumour, and I had no hesitation in operating without further evidence, and removed a considerable villous papilloma.

The appearance of long villous processes in the urine-*débris*, I take to be decisive. They are in my experience never seen as normal products, or in the absence of such diseased growths in the bladder.

Another suspicious product is the appearance of a mass of adhering fusiform or spindle-shaped cells of large size; some of them lengthened out into fibres, and when seen more or less regularly in the urine of a patient, with other symptoms, should arouse grave suspicions; but these cells are not pathognomonic like the villous. Large round cells of various kinds, often young epithelium, are of course not sufficiently characteristic by themselves. Then I have met with large, soft-looking fibres with nuclei on them, as if recently developed from cells, and believed them to be significant of growth of some kind (associated with tumour symptoms and history), have explored the bladder and found nothing! This I did in two cases, and it is worthy of remark that in both instances the patient was much benefited by the operation.

4. *The co-existence of calculus with tumour is to be noted.*—In two cases at least, there had been previous formation, in the one of an oxalate of lime calculus, and in the other of several small uric acid calculi, producing continued irritation of the bladder for some time. In both instances lithotrity had been performed recently. In a third case, that of a woman, a large calculus was found in the kidney, the symptoms of which had no doubt masked the existence of the vesical tumour; and had, before I saw her, been regarded as the cause of the hæmaturia. These facts at all events lend some support to the theory that papilloma may sometimes arise from local sources of irritation affecting a mucous membrane.

There is one important point still to be referred to, viz. what is the proportion of instances regarding tumours of the bladder generally, in which we may expect to find the physical condition of the growth such as will admit of complete removal, or almost complete removal? I permit myself to say "almost complete" removal, because I am satisfied that with non-malignant products, like papilloma for example, a complete ablation of the growth is not absolutely essential to success. I am quite certain that I have removed the greater part, but not the whole of the

tumour in two cases at least, in which there has been no sign of reappearance for upwards of a year. I feel little doubt that when the chief mass of a papillomatous growth has been nipped off by means of forceps in or near to the base, it is impossible to affirm that all the irregular formation has been taken away; on the contrary it is reasonable to suppose that some portion must always have been left. I believe that cicatrisation takes place, and that by degrees this process leads to solidification of the tissues at the point at which evulsion was made.

I have had in one case the unusual advantage of being able to place my finger on the very site of an evulsion performed nine months before, and I then found no reappearance of growth so far as my sense of touch enabled me to judge. In the case of malignant tumours of course no attempt would be made to remove such growths; for in no case would there be the smallest hope of being able to remove the diseased formation entire. Nor, again, were it conceivable that we could accomplish this, is there any reason to believe that advantage would accrue to the patient thereby.

I have recently made a careful examination of most of the specimens of tumour of the bladder preserved in the museums of London, and I have found about a hundred preparations, relative to which I could determine several important questions. Of these I regard about forty as malignant, and about sixty as examples of non-malignant growth; that is, specimens of what may be considered papilloma and innocent forms of villus. Of these sixty preparations, not less than thirty-five appear to me to be removable by operation. It should be remarked that many examples are labelled "cancer" which cannot be so regarded; a large portion undoubtedly are so; several are probably epithelioma; but the allegation of malignancy must not be accepted in a great number in which it is made. It is, then, an instructive and striking fact that at least one half of the simple growths can be removed with a good prospect of success. I believe the

proportion is greater than this, but I have been desirous to understate it rather than the contrary.

I shall now only add that I have performed the operation described for the purpose of ascertaining the cause of severe and long-standing symptoms when obscure, in 27 cases altogether; 21 of them occurring within the last twelve months. Among them I found 12 cases of vesical tumour, and removed it wholly or partially in 10. In 2 only I declined the attempt. I cannot therefore doubt that the prevalence of these affections is greater than it has been customary to believe it to be; and that the victims of it hitherto have, after much unavailing treatment, both surgical and medical, slowly bled to death, under the impression that the source of the hæmorrhage was renal, or at all events beyond the reach of any aid from art.

Appendix.

Case 1 (report of Mr. Stanley Boyd).—The growth consisted of three chief masses of roughly spherical form, one half to three quarters of an inch in diameter, and having short narrow pedicles, and of two or three small sessile masses of similar shape. Bits of surrounding, apparently normal mucous membrane had also been removed. All the nodules were velvety on account of the projection everywhere from their surfaces, of thin folds, and embranched, somewhat club-shaped processes, one sixteenth of an inch or less in length. Under the microscope these processes consisted almost entirely of one or two very thin-walled vascular loops of wide calibre, but some showed a good deal of round-celled infiltration. They were covered by a thick layer of epithelium, the component cells of which were columnar, very long and narrow; the epithelium stripped off with the greatest ease. The mass of the tumour consisted of rather loose connective tissue, containing here and there small collections of round cells. No glandular structure was seen.

Vessels were large, numerous, and provided with stout coats in the body of the growth, but towards the surface large numbers of the same wide, thin-walled vessels were seen as were noticed in the processes on the surface. They had no muscular fibres in their walls. The surface of the growth between the processes is covered by epithelium similar to that on the villi. At the base of the nodule examined some bundles of the involuntary muscular layer of the bladder were seen, but no such tissue existed in the growth.

Case 3 (report of Mr. Stanley Boyd).—The tumour consists of several rounded or oval, more or less pedunculated masses, varying in size from that of a chestnut downwards. Apparently they were connected with the submucous tissue, but it is impossible to be certain that they did not extend more deeply. On the surface the growths are velvety, frequently encrustated with phosphates; no processes were seen until a section was mounted, then several very short ones were found breaking the uniformity of the surface.

Microscopically there is a stroma of connective tissue with bands of a denser nature running in different directions. Cells are everywhere numerous, and in some parts the infiltration is so dense that one would have been anxious—had the patient lived—lest recurrence should increase the grounds for fearing that the tumour was a fibro-sarcoma. As, however, epithelium is present in many places, and considerable areas of loose connective tissue (like that of a nasal polypus) are seen, whilst the vessels have everywhere distinct, *i.e.* not formed by the cells of the growth, though thin walls, it seems most probable that the tumour was a loose fibroma.

Case 4 (report by Dr. H. Gibbes).—This tumour consists of a villous outgrowth, made up of processes which branch and finally end in long filiform villi; each villus is composed of a central portion, consisting of white fibrous tissue and connective-tissue corpuscles, with a large amount of non-striped muscle. This muscle is continued

for some distance in distinct bands, but does not extend into the filiform processes, which latter are composed of fibrous tissue and small round cells. In each villus runs a blood vessel, which branches and is continued into the filiform processes up to the epithelium. All parts of the growth are covered with a stratified layer of columnar epithelium, in which many of the cells are distended with mucus. In some parts of the villi there are crypts, lined with stratified columnar epithelium, and in places these may be seen filled with loose cells and amorphous urinary products, and the cells are undergoing fatty degeneration. The whole growth is of a firm consistence and not very vascular. The cells on the surface are large, and contain a large nucleus which shows the intra nuclear network very plainly. The cells are set in a distinct basement membrane.

Case 5 (Report by Mr. Shattock).—The portions of growth are thinly encrusted with white phosphatic material, and on section are much blood-stained from hæmorrhage into their substance. On teasing these out a low simple papillary construction of surface, with a corresponding vertical arrangement of engorged vessels is recognisable.

Microscopic sections appear much infiltrated with blood. Large portions of the section are constructed of a highly-irregular meshwork of homogeneous (fibrinous) substance enclosing pigment granules, which also extend amongst the proper elements of the tumour; the latter consist of somewhat large nucleated spherical cells. The tumour may be named a "sarcomatous papilloma," and be classed in the same category as the "soft warts."

Case 6 (report by Dr. Gibbes).—This tumour consists of large masses of stratified squamous epithelium, in the centres of which are many epithelial nests. In the middle of these masses runs a small quantity of fibrous tissue, which is infiltrated with small round cells, and a small blood-vessel is present in the centre. These small round cells are found throughout the whole growth in

large numbers, and from their varied shapes they are evidently in an active state of division. The nuclei of the squamous cells themselves are also in many places germinating, and have formed large cells filled with the products of the division of the original nuclei. This is seen in many parts of the superficial epithelium, and in places cysts seem to have been formed, which are now filled with a fibrinous mass. There are a number of blood-vessels running through the fibrous tissue. The tumour has all the usual characters of an epithelioma.

Case 7 (report by Dr. Gibbes).—This growth consists of a number of delicate filiform processes. Each of these consists of a central stalk from which branch several secondary processes. They are formed of a very delicate connective-tissue in the centre, which is infiltrated with small round cells, and they are covered with a stratified layer of columnar epithelium, which resembles that of the normal bladder. This epithelium is set on a nucleated basement membrane; a large blood-vessel enters at the base of each villus, and branches as it goes on until it finally breaks up into a network of capillaries, which lie directly under the basement membrane. In some places these capillaries may be seen to have ruptured on to the external surface. The whole growth is very vascular. Many of the columnar epithelial cells are distended with mucus. In some of the villi there are crypts lined with columnar epithelium similar to that on the surface.

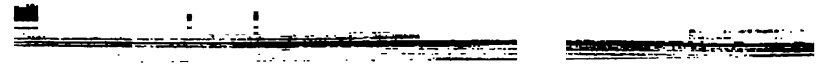
Case 8 (report by Dr. Gibbes).—The growth consists of a number of very delicate filiform processes covered with layers of columnar epithelium cells. Each process is formed of a fine connective-tissue, infiltrated with small round cells; the epithelium is arranged in several layers on the surface. In each process are a number of capillary blood-vessels, which run towards the surface and branch directly beneath the epithelium.

The growth is very vascular, and in some places the vessels are much distended with blood. The vessels are ruptured also in several instances, and masses of blood

are seen on the surface; whether this was done in the act of removing the growth it is impossible to say with certainty. There is nothing malignant about the tumour; it is entirely composed of normal bladder tissue.

Case 12 (report by Dr. Gibbes).—This tumour appears to be an hypertrophy of the sub-mucous coat of the bladder.

The muscle appears normal, but the tissue inside it is composed of dense bands of fibrous tissue, which have a macerated or soddened appearance, as if there had been great œdema into them; nearer the epithelium the fibrous tissue is much finer, and directly under the epithelium it has a reticulated appearance, exactly resembling granulation-tissue in the bottom of a healing wound. The epithelium on the surface resembles that of the normal bladder in every respect. The blood-vessels in the depths have very thick walls, and are surrounded with round cells in some places. The capillaries run directly to the surface, generally without branching, and are there ruptured in many places. They are also ruptured in some parts of the deeper tissue, and there are many spots of extravasated blood. There are collections of round cells in some places, resembling lymphoid tissue, and these are arranged in round or oval masses. There are no villous growths in that portion of the tumour examined.



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